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THE SUBCLAVIAN ARTERIES: ROENTGEN STUDY IN HEALTH AND DISEASE*

By NATHAN POKER, M.D., NATHANIEL FINBY, M.D., *and* ISRAEL STEINBERG, M.D.
NEW YORK, NEW YORK

TWENTY years experience with roentgen visualization of the cardiovascular system has resulted in the accumulation of considerable data regarding the subclavian arteries in health and disease. This paper discusses the methods of roentgen examination, describes the normal roentgen appearance, and demonstrates the significance and variation in disease of the subclavian arteries.

NORMAL ANATOMY

The right subclavian artery arises from the innominate artery behind the sternoclavicular articulation. The left subclavian artery arises from the arch of the aorta behind the left common carotid artery, forming the third branch of the brachiocephalic arteries. Both subclavian arteries course upward and laterally in the superior mediastinum, arch over the dome of the pleura and pass behind the anterior scalene muscle. Thence they pass behind the clavicle to the lateral borders of the first ribs where they continue as the axillary arteries. For descriptive purposes, the anterior scalene muscle divides the subclavian artery into three parts: first, from the origin to the medial border of the scalenus anticus muscle; secondly, behind the scalenus anticus muscle; and thirdly, from the lateral border of the scalenus anticus muscle to the outer border of the first rib.

In its first part, the right subclavian artery is adjacent to the vagus, the cardiac sympathetic and the right recurrent laryngeal nerves, the internal jugular and vertebral veins and the pleura at the apex of the lung. In its first part, the left subclavian artery is related to the vagus, phrenic, left recurrent laryngeal and cardiac nerves, the left common carotid artery, the esophagus, the thoracic duct, the trachea and the pleura at the apex of the left lung. Both subclavian arteries in their second parts are adjacent to the brachial plexus (above), the pleura (below), and the anterior and middle scalene muscles. In their third parts, they are adjacent to the scalenus muscle, the trunks of the brachial plexus, the first ribs (posteriorly) and the clavicle and deep cervical fascia (anteriorly). This is the most superficial portion of the subclavian

*From the Department of Radiology, The New York Hospital-Cornell Medical Center, New York, New York.
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arteries. Important branches are the vertebral, internal mammary, thyrocervical, costocervical, and transverse cervical arteries. These participate in the collateral circulation when the subclavian artery is obstructed.[47]

ROENTGEN ANATOMY

Roentgenographically, the subclavian arteries are invisible in frontal and lateral views. However, the left subclavian artery can be visualized in the left anterior oblique view, and Parkinson and Bedford[52] have emphasized the value of this view during cardiac roentgenoscopy, not only for the demonstration of the aortic window, but also for the clear space above the aortic arch which they termed, "the aortic triangle." The base of this triangle is formed by the aortic arch, its left side by the spine, and its right side by the left subclavian artery (Fig. 1). These relationships were established by postmortem roentgen studies using radiopaque markers on the left subclavian artery. Parkinson and Bedford also noted decrease in the size of the aortic triangle in conditions which elevate the aortic arch (arteriosclerosis, hypertension and syphilitic aneurysms). Increase in the size of the aortic triangle occurs in the diseases which lower the aortic arch (emphysema and asthenic habitus). They also described decrease in the aortic triangle when the left subclavian artery was displaced by retrosternal goiters and other thoracic tumors.

Following angiocardiography, all the vascular trunks arising from the aortic arch are clearly opacified.[75] The right subclavian artery is seen in its entirety in frontal projection (Fig. 2), while the first portion of the left subclavian artery is best visualized in the left anterior oblique view (Fig. 1). This is because of the oblique course of the aortic arch. Subclavian artery opacification may also be obtained by direct puncture of the aortic arch or by catheterization of the radial artery with injection of contrast material into the aorta (retrograde arteriography) (Fig. 3). The latter is especially valuable for outlining obstructive lesions of the subclavian artery.[59]

Angiocardiographic Measurement of the Subclavian Arteries. The length and caliber of the right and left subclavian arteries opacified by the method of angiocardiogra-

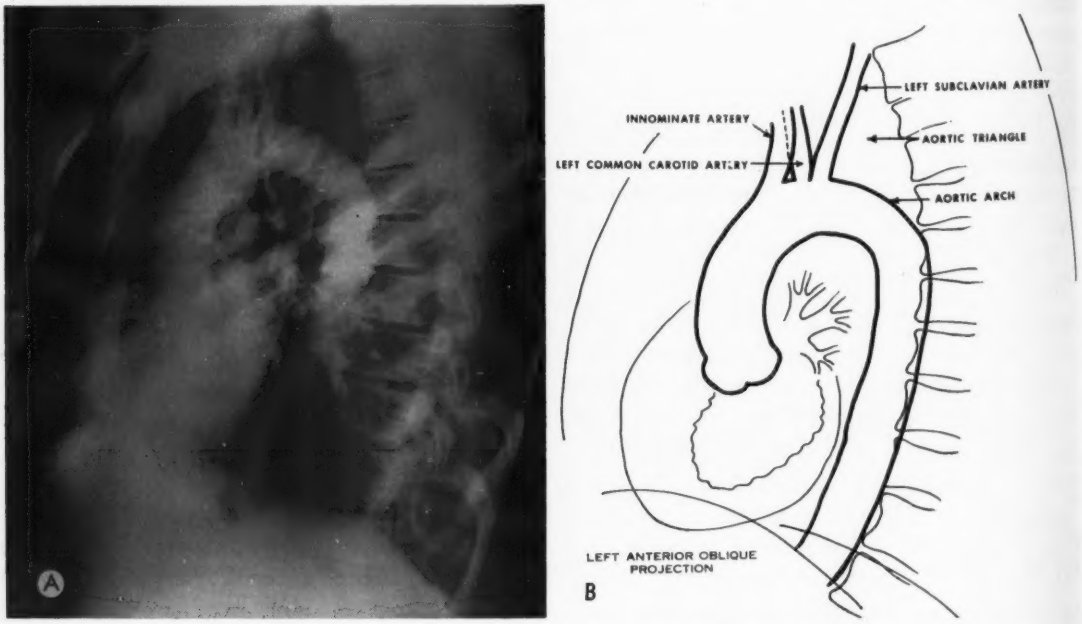

Fig. 1. The normal left subclavian artery and aortic triangle. (*A*) Left anterior oblique angiocardiogram. (*B*) Tracing of *A*.

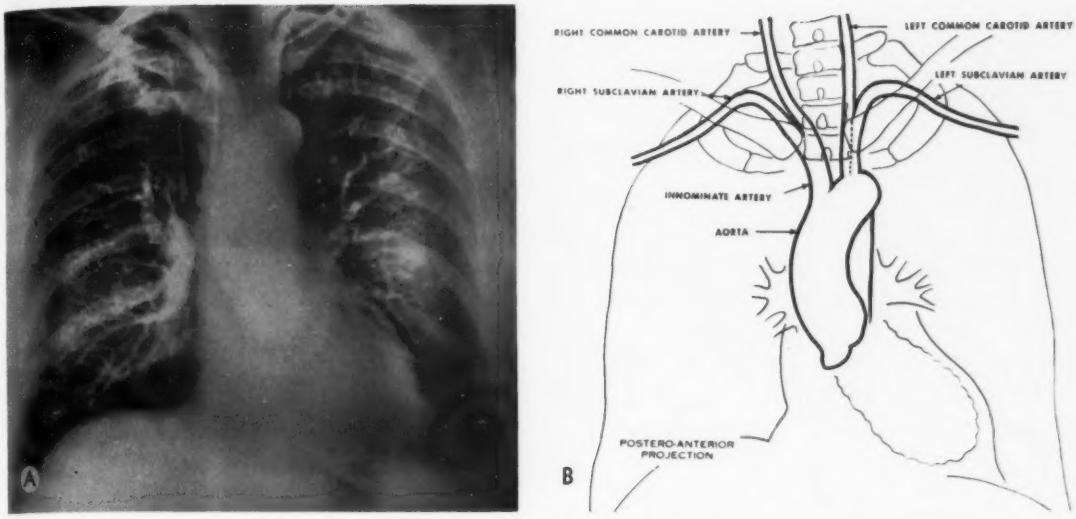

FIG. 2. The normal subclavian arteries. (*A*) Frontal angiocardiogram showing the normal right subclavian artery. (*B*) Tracing of *A*.

phy[14] were measured in patients of both sexes and various ages in normal and pathologic states (Table 1). The left anterior oblique projection was used for measurement of the left subclavian artery while the frontal projection was utilized for the right subclavian artery (Fig. 4). The average normal length of the left subclavian artery was 5.0 cm.; the right subclavian artery, 6.1 cm. The average normal caliber of the right subclavian artery at its summit was 0.6 cm.; the average normal caliber of the left subclavian artery at its estimated midpoint was 0.9 cm. (Table 1). The ages of the patients ranged from fourteen to sixty-eight years. The variation of subclavian artery length with age is shown in Figure 5.

Numerous errors are inherent in this method of measurement. Differences in positioning of the patient, roentgen distortion, variation in age, and difficulties in measuring poorly opacified vessels undoubtedly introduce errors. These measurements, therefore, are approximations rather than absolute. However, a review of the literature shows no other tabulation. Furthermore, anatomic texts give only rough estimates of length and no caliber measurements.[47]

TABLE I

ANGIOCARDIOGRAPHIC MEASUREMENT OF THE SUBCLAVIAN ARTERIES

	Age Range (yr.)	Left Subclavian Artery (Left Anterior Oblique Projection) Caliber (cm.)					Right Subclavian Artery (Posteroanterior Projection) Caliber (cm.)*					Total Cases Studied
		No. of Cases	Length (cm.)	Origin	Mid-portion	Termination	No. of Cases	Length (cm.)	Origin	Summit	Termination	
Normal (without cardiovascular disease)	14–68	77	5.0	1.0	0.9	0.8	29	6.1	0.6	0.6	0.5	100
Cardiovascular syphilis	38–71	57	4.2	1.0	0.9	0.9	4	5.8	0.8	0.7	0.7	58
Hypertensive and arteriosclerotic heart disease	27–69	20	4.0	1.0	0.9	0.8	9	5.7	0.6	0.6	0.5	26
Congenital heart disease	14–66	29	4.1	0.9	0.9	0.8	2	4.2	0.5	0.5	0.5	31
Thoracic tumors	14–58	8	4.1	0.9	0.8	0.8	13	5.5	0.6	0.5	0.5	20

* See Figure 4.

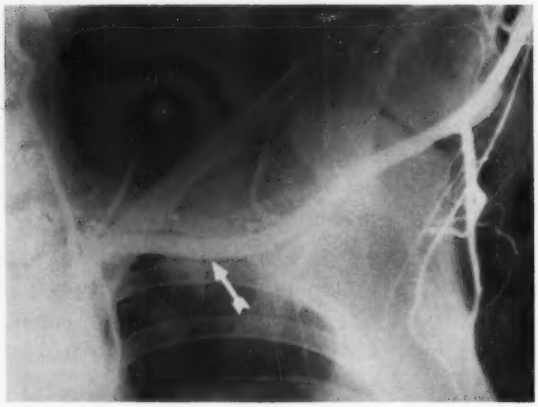

FIG. 3. Arteriogram showing the normal left subclavian artery. Arrow points to termination of third part of left subclavian artery at outer border of the first rib.

Measurements of the subclavian arteries were also made in four disease groups: syphilitic, arterioslcerotic and hypertensive, congenital cardiovascular disease, and thoracic tumors. These measurements are also recorded in Table 1. The studies indicate that the subclavian arteries are shorter in arteriosclerotic and hypertensive cardiovascular disease (Fig. 6). This is undoubtedly associated with the elevation, elongation, and tortuosity of the unfolded aortic arch. In cardiovascular syphilis (Fig. 7 and 8), the decrease in length of the subclavian arteries is of lesser extent and is related to aortic length and position, and perhaps to the extension of the pathologic process from the aorta. In congenital heart disease (chiefly coarctation of the aorta), an apparent decrease in length of the subclavian arteries results from deformities of the great vessels. Finally, the tumor group, which comprises intrathoracic masses in the superior mediastinum, shows apparent decreased length of the subclavian arteries due to local displacement or distortion.

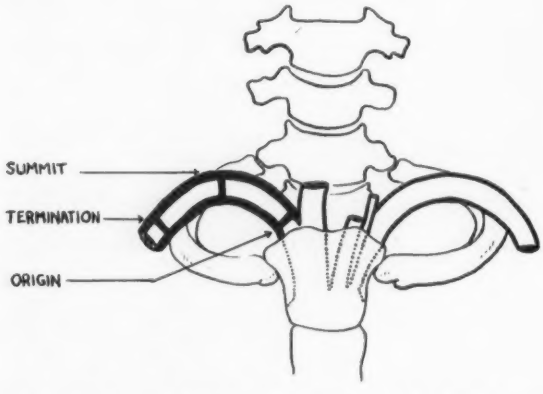

FIG. 4. Schematic diagram showing the sites of angiocardiographic measurement of the right subclavian artery.

THE SUBCLAVIAN ARTERIES IN DISEASE

The subclavian arteries are involved by the same pathologic processes which afflict other portions of the vascular system; namely, arteritis, arteriosclerosis, aneurysm, congenital anomalies, arteriovenous fistulas, neoplasms, thrombosis and trauma.

The subclavian arteries are of clinical importance in many congenital and acquired conditions. For instance, in the Blalock-

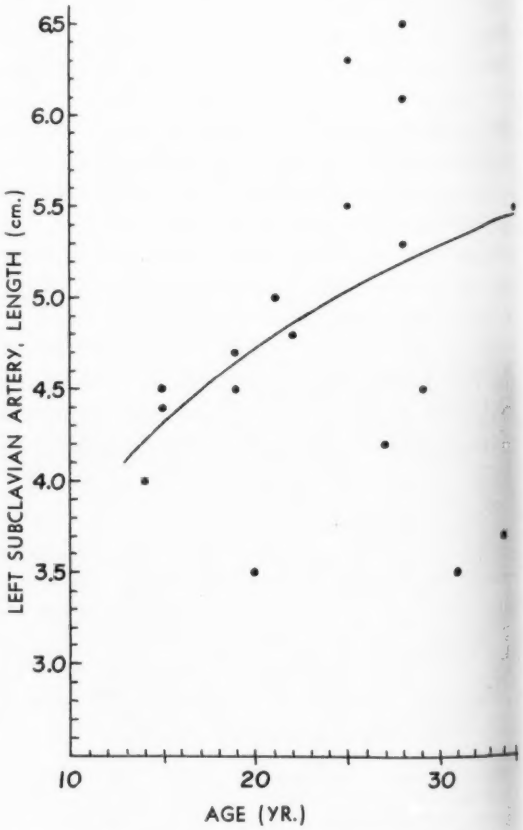

FIG. 5. Scatter diagram relates age of patient to length of left subclavian artery. Good correlation is found between the ages of fourteen to thirty-five years.

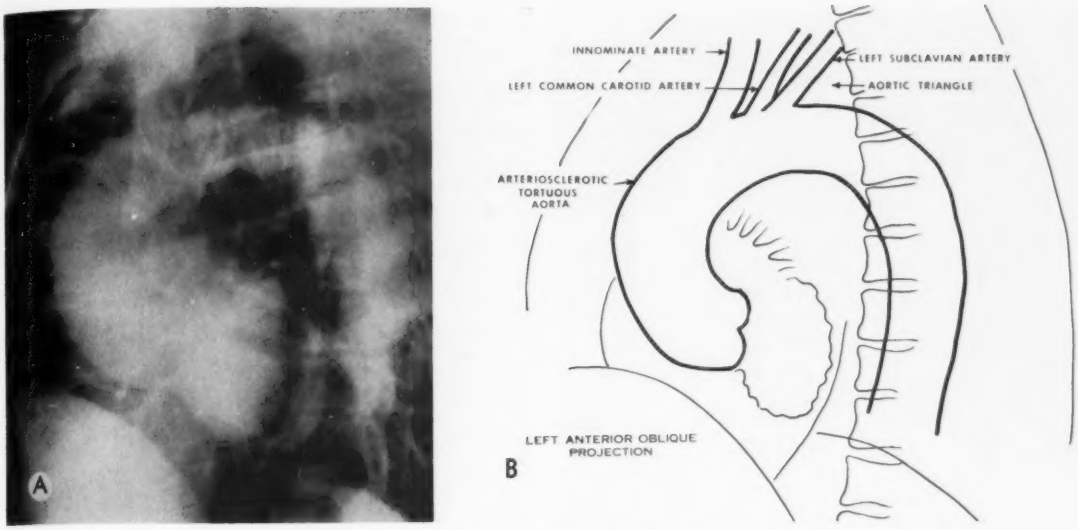

Fig. 6. The left subclavian artery in arteriosclerosis of the aorta. (*A*) Left anterior oblique angiocardiogram. (*B*) Tracing of *A*. Note the decreased size of aortic triangle.

Taussig operation, information regarding the caliber and length of the subclavian vessels is valuable preoperatively. The subclavian arteries are frequently involved in the neurovascular syndromes of the thoracic inlet and shoulder girdle (cervical rib, scalenus anticus, costoclavicular and hyperabduction syndromes). Congenital anomalies as well as degenerative, traumatic and neoplastic diseases may involve the subclavian arteries.

Table II summarizes our experience and presents a classification.

I. CONGENITAL ANOMALIES OF THE SUBCLAVIAN ARTERIES

a. Anomalous Origin.

Variations in the origin of the arteries arising from the aortic arch have long been recognized at necropsy. Instead of the usual three brachiocephalic arteries, as many as five or as few as two main trunks have been

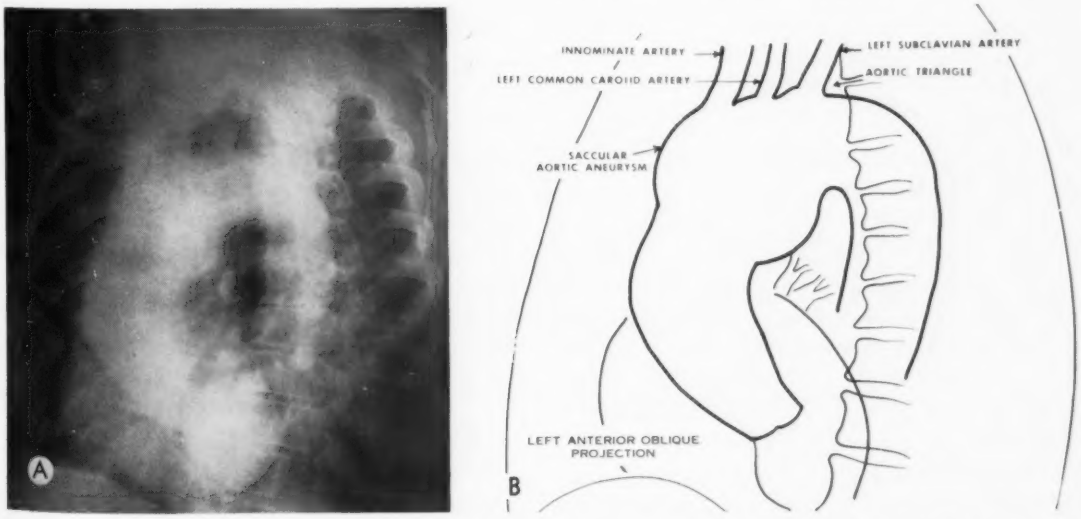

Fig. 7. The left subclavian artery in syphilitic aneurysm of the aortic arch. (*A*) Left oblique angiocardiogram. (*B*) Tracing of *A*. Note marked decrease in size of aortic triangle.

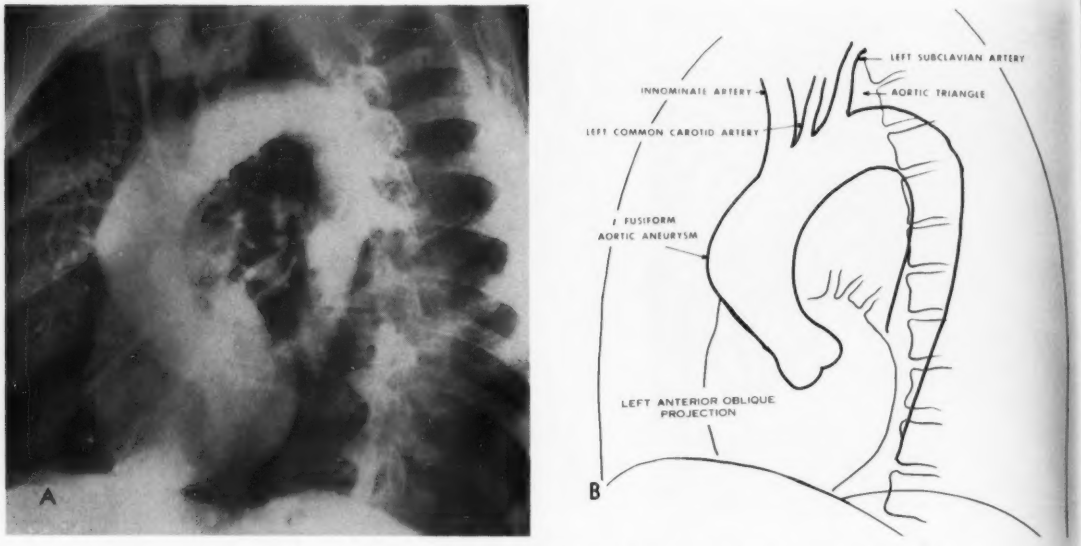

FIG. 8. The left subclavian artery in syphilitic aortitis. (*A*) Left oblique angiocardiogram. (*B*) Tracing of *A*. Again, note decrease in aortic triangle.

described (Fig. 9 and 10). The individual origin of the vessels may also vary; the most common variant is an aberrant right subclavian artery arising as the last, or fourth branch, of the aortic arch (Fig. 11). The origin of the right subclavian artery as a first branch is uncommon. Rarely, the right subclavian artery arises as the second or third branch. When it originates as the fourth branch, it courses to the right and is retroesophageal in 80 per cent of the cases, sometimes causing dysphagia (dysphagia lusoria). In 15 per cent, it passes between the trachea and esophagus and in 5 per cent it is anterior to the trachea.[53]

Anomalous subclavian arteries have been

TABLE II

CLASSIFICATION OF DISEASES OF THE SUBCLAVIAN ARTERIES

I. Congenital
  a. Anomalous origin
  b. Congenital arteriovenous fistula and aneurysm
  c. Coarctation and pseudocoarctation
II. Acquired
  a. Thoracic inlet syndrome
  b. Buckling
  c. Aneurysm
    (1) due to cervical rib
    (2) due to syphilis
    (3) due to arteriosclerosis
    (4) due to trauma
  d. Arteriovenous fistula
  e. Occlusion
    (1) aortic arch syndrome (pulseless disease)
    (2) due to cervical rib
    (3) due to trauma
  f. Neoplasms involving the subclavian arteries
III. Miscellaneous
  a. Interruption of the aortic arch
  b. Occlusion following Blalock-Taussig operation
  c. Thoracoplasty and kyphoscoliosis

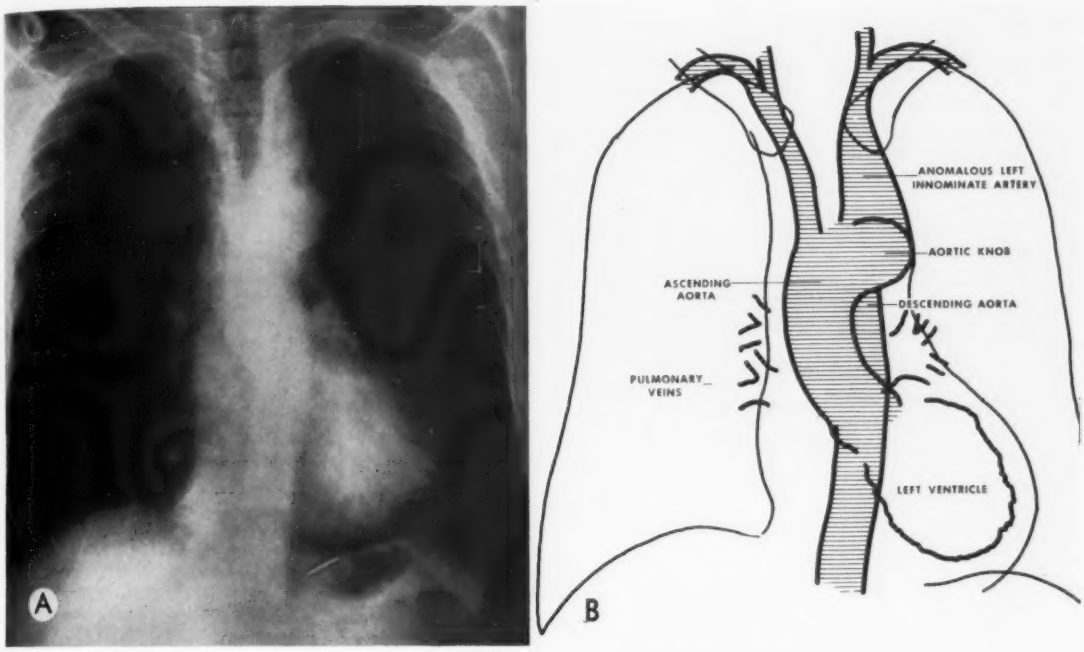

FIG. 9. Common origin of left subclavian and carotid arteries ("left innominate artery"). (*A*) Frontal angiocardiogram. (*B*) Tracing of *A*.

reported in the tetralogy of Fallot,[81] pulmonic stenosis,[2] patent ductus arteriosus,[5] and Eisenmenger's complex.[4] The most common association is with tetralogy of Fallot. Bahnson and Blalock[2] report 36 cases of aberrant right or left subclavian arteries in 841 patients.

b. Congenital Subclavian Arteriovenous Fistula and Aneurysm.

Congenital arteriovenous fistulas of the subclavian artery (Fig. 12) are rare. In a review, Callander[9] found only 3 among 447 cases. Congenital arteriovenous fistulas have also been reported by Reid,[60] Pemberton and Saint,[54] and Perasado and Kyllonen.[55] Congenital aneurysm of the subclavian arteries is also rare. Recently bilateral aneurysms were reported in a fourteen year old boy.[56]

c. The Subclavian Arteries in Coarctation of the Aorta.

Dilatation of brachiocephalic vessels proximal to aortic coarctation is frequent (Fig. 13). Indeed, dilatation and pulsation of the left subclavian artery with widening of the left superior mediastinal shadow have become diagnostic signs.[70] Atresia or stenosis of either subclavian artery accompanying aortic coarctation is, on the other hand, uncommon. This condition should be suspected when there are differences in the blood pressures of the upper extremities. In a review of 170 cases of coarctation of the aorta, 10 had unequal blood pressures in the upper extremities.[36] In 9 of the 10 cases the pressure was higher in the right arm and is explained by the involvement of the left subclavian artery in the aortic coarctation. This often caused a unique clinical syndrome in children: hypertrophy of the right side of the chest and of the right upper extremity with absent pulse in the left arm.[66] Right subclavian artery atresia accompanying coarctation is exceedingly rare.[41,50]

Pseudocoarctation[71] is an abnormality of the aorta consisting of deformity or kinking of the aortic arch without occlusion of the aortic lumen. Patients with pseudocoarctation are usually asymptomatic and are referred because of pulsatile superior mediastinal masses. The blood pressures of the

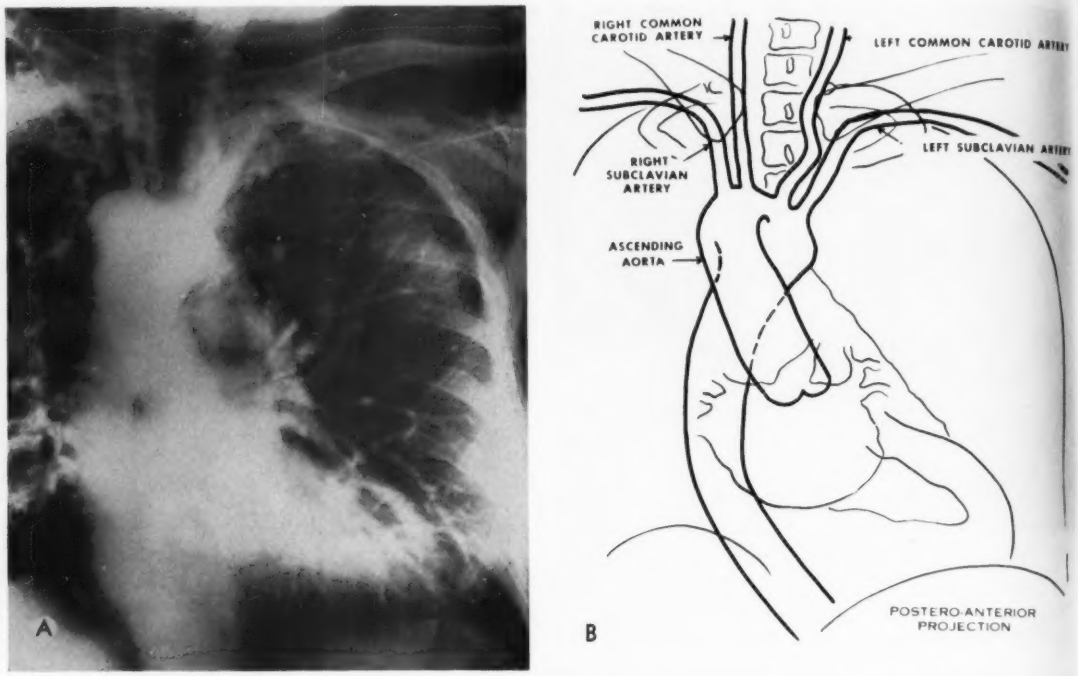

Fig. 10. Separate origin of right subclavian artery in patient with right aortic arch and left aortic diverticulum. (*A*) Frontal angiocardiogram. (*B*) Tracing of *A*.

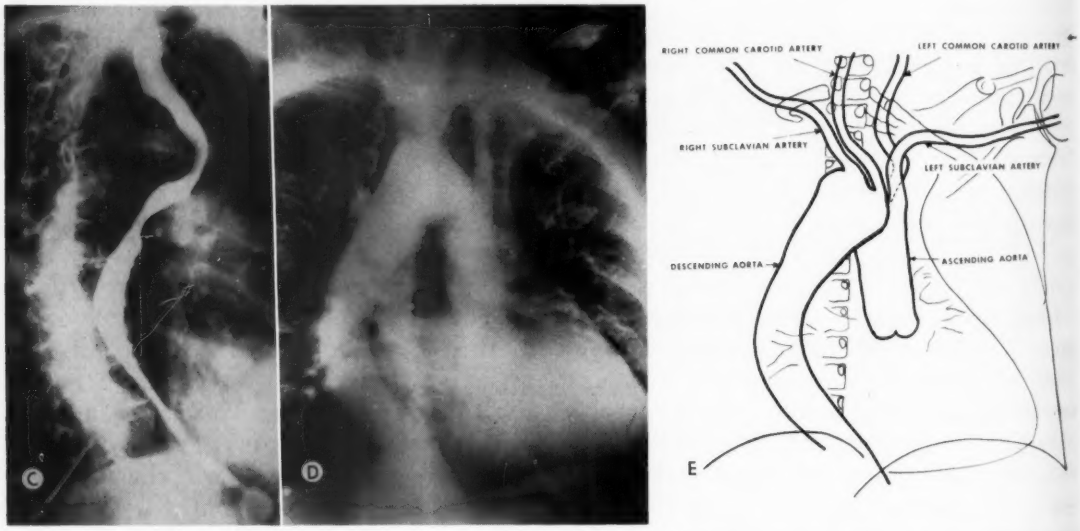

Fig. 10. (*C*) Characteristic esophagram of right aortic arch. (*D*) Right anterior oblique angiocardiogram demonstrating the cause of the esophageal deviation. (*E*) Tracing of *D*.

lower extremities are unaltered; angiocardiography discloses no collateral aortic arch circulation. Associated changes in the left subclavian artery consist of striking dilatation, a more distal origin from the aortic arch, and a longer intrathoracic course (Fig. 14).

II. Acquired Disease of the Subclavian Arteries

a. The Subclavian Arteries in the Thoracic Inlet Syndrome.

Compression of the neural and vascular structures at the thoracic intlet is a fre-

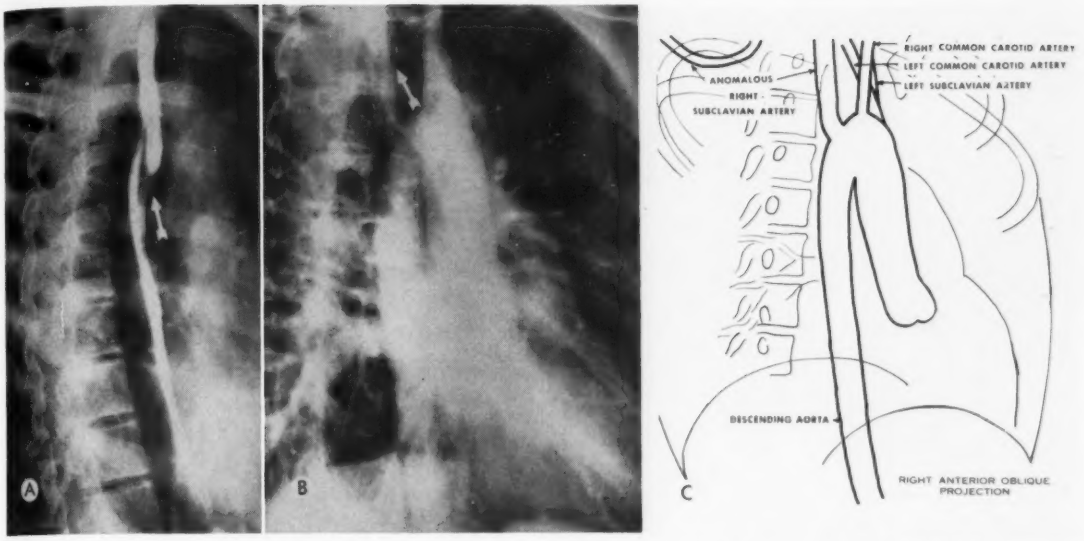

FIG. 11. Anomalous origin of right subclavian artery (arrow) from distal portion of aortic arch causing dysphagia lusoria in a fourteen year old girl. (*A*) Right anterior oblique esophagram showing characteristic indentation (arrow) by anomalous right subclavian artery. (*B*) Right anterior oblique angiocardiogram. (*C*) Tracing of *B*.

quent cause of pain in the upper extremity. The proper therapy depends upon evaluation of all the factors that can produce compression of the subclavian vessels and brachial plexus. Early reports stressed the importance of structural abnormalities such as cervical ribs. Hence the term "cervical rib syndrome." Subsequently, when it became apparent that the neurovascular syndrome of the shoulder girdle could occur without osseous malformations and could be due to torsion, swelling or hypertrophy of the scalenus muscles, the term "scalenus anticus syndrome" became popular.[48] Later, investigators further observed that symptoms independent of organic or structural change might be due to unusual positions of the upper extremity, especially during the patient's occupation or sleeping posture. Thus, Falconer and Weddell[19] in 1943 demonstrated compression of the subclavian vessels and downward bracing of the shoulder in soldiers carrying heavy packs. They termed this the "costoclavicular syndrome." Soon after, Wright[84] in 1945 showed severe vascular and neurologic changes in patients holding the upper extremity in prolonged extreme abduction, as in sleeping with the arm overhead, and termed this the "hyperabduction syndrome." It is obvious that there is a distinction between the hyperabduction syndrome aggravated by postures with the extremity overhead and the scalenus anticus and costoclavicular syndromes, which are made worse by downward and backward position of the arm. The differential diagnosis of these syndromes can only be determined by careful clinical study. Beyer and Wright[3] have masterfully outlined the clinical maneuvers necessary for accurate diagnosis.

Roentgenography plays a significant role in determining the type of thoracic inlet syndrome, especially by indicating the presence or absence of cervical ribs, elongated transverse spinal processes and bony exostoses. Opacification studies can also demonstrate impingement on the subclavian vessels (Fig. 15). Retrograde arteriography has been successfully employed for this purpose.[49] Angiocardiography may also be of value for delineation of subclavian artery abnormalities such as stenosis, thrombosis, or aneurysm. Lord[40] has emphasized that thorough clinical appraisal and precise anatomic localization of the lesion are desirable prior to surgery.

FIG. 12. Arteriogram showing congenital arteriovenous fistula of the left subclavian, axillary and brachial arteries.

Since the surgeon must decide whether cervical costectomy, anterior scalenectomy, or claviculectomy will best alleviate the neurovascular compression, it is obvious that careful preoperative clinical and roentgen evaluation is essential.

b. Buckling of the Subclavian Arteries.

Seven patients had a pulsatile swelling in the right supraclavicular area due to buckling of the right subclavian artery associated with hypertension and arteriosclerosis of the aorta (Table III). In 2, this was the primary cause for admission but, in the remainder, buckling was found by angiocardiography. The ages of the 7 patients ranged from forty-one years to seventy-five years; there were 6 females and 1 male. Duration of symptoms was from ten months to eleven years. Four of the 7 patients had severe hypertension. Angiocardiography (Fig. 16) demonstrated that the right supraclavicular pulsatile masses were due to tortuosity and buckling of the subclavian artery, apparently related to hypertension and arteriosclerosis of the aorta. In 2 cases there was concomitant buckling of the innominate artery with pronounced widening of the right superior mediastinum. Although buckling of the innominate artery has been well documented,[30,31] elongation and tortuosity of the subclavian arteries have been reported only recently.[33] Angiocardiography permits differentiation of supraclavicular masses and

FIG. 13. Left anterior oblique angiocardiogram showing dilatation of the left subclavian artery in coarctation of the aorta. Arrows point to collateral circulation.

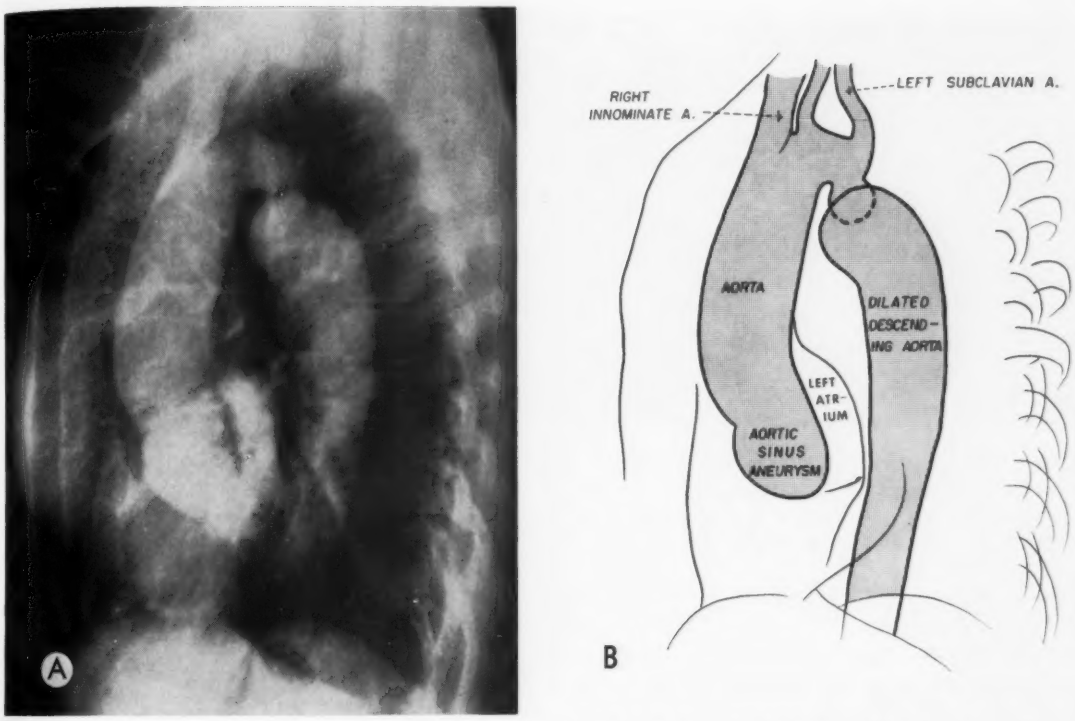

Fig. 14. The left subclavian artery in pseudocoarctation. (*A*) Left anterior oblique angiocardiogram. Note absence of collaterals. (*B*) Tracing of *A*. By permission of *The British Heart Journal*.[71]

Table III

BUCKLING OF THE RIGHT SUBCLAVIAN ARTERY ASSOCIATED WITH HYPERTENSION AND AORTIC ARTERIOSCLEROSIS

New York Hospital No.	Age	Sex	History	Physical Examination	Blood Pressure mm. Hg	Angiocardiographic Findings
614274	54	F	Pulsation and swelling in right side of neck, progressive for 11 years	Four by three cm. pulsatile mass in right supraclavicular area	195/110	Retrograde arteriography showed marked tortuosity of second portion of right subclavian artery
730580	64	M	Swelling in neck for 10 months	Diffuse, pulsatile, nontender swelling in right sternoclavicular area	160/ 90	Tortuous second part of the right subclavian artery
642282	41	F	Hypertension for 10 years. Asymptomatic mass in neck	Pulsatile mass in neck. Artery dilated with thrill and systolic murmur	245/128	Tortuous right subclavian and common carotid arteries. Buckled innominate artery
514288	56	F	No symptoms referable to mass in neck	Pulsatile mass in right side of neck 2×4 cm.	168/ 96	Tortuous right subclavian and common carotid arteries with marked buckling far above clavicle
575299	62	F	Constant throbbing in right side of neck for 7 years; worse 2 years	Right carotid artery pulsation easily visible and palpable	140/ 80	Moderate buckling of right subclavian and innominate arteries
719910	61	F	Prominent right superior mediastinal mass	Cervical mass not palpable. Marked pulsations in right superior mediastinum roentgenoscopically	200/140	Buckling of second portion of the right subclavian artery (Fig. 16). Innominate artery dilated (15 mm.)
592687	75	F	No symptoms referable to mass in neck	Pulsatile mass in neck. Loud to-and-fro murmur	210/120	Tortuosity of the right subclavian artery

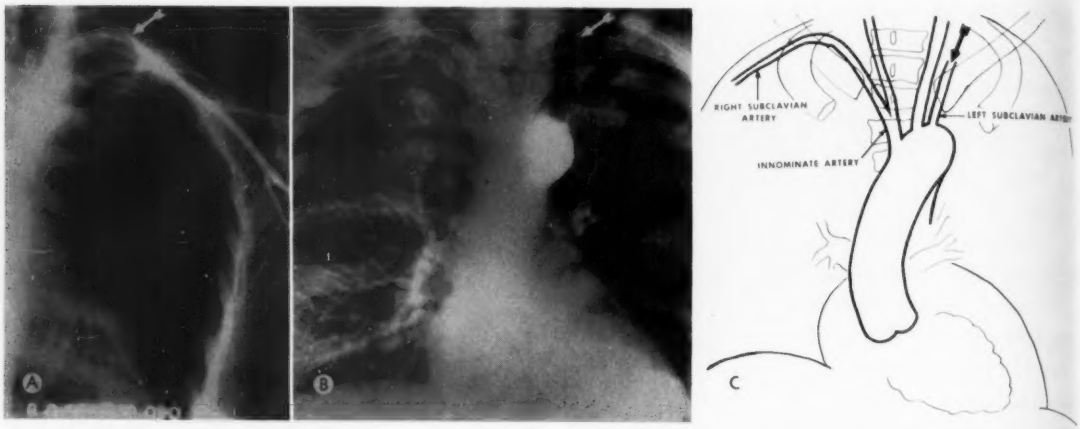

FIG. 15. Scalenus anticus syndrome in a fifty-six year old woman with numbness, tingling and absent radial pulse on the left. (*A*) Retrograde arteriogram showing occluded second portion of the left subclavian artery (arrow). (*B*) Frontal angiocardiogram also reveals point of occlusion with absent filling of distal portion of left subclavian artery. Arrow points to site of occlusion. (*C*) Tracing of *B*.

subclavian arteriovenous fistula and aneurysm.

c. Aneurysm of the Subclavian Arteries.

Aneurysms of the subclavian arteries are uncommon. Their diagnosis may establish the need for corrective therapy and thereby avoid rupture, occlusion and erosion of contiguous structures. It is important to know the etiology of subclavian artery aneurysms because the tendency to rupture varies in the different types. The following is a convenient classification of the subclavian artery aneurysms.

(1) Subclavian Artery Aneurysms Associated with Cervical Ribs. Aneurysms of the subclavian artery secondary to cervical ribs have been reported with differing frequency. Deitmar[13] in 1907 reported 5 cases. In 1916, Halsted and Reid[26] found 27

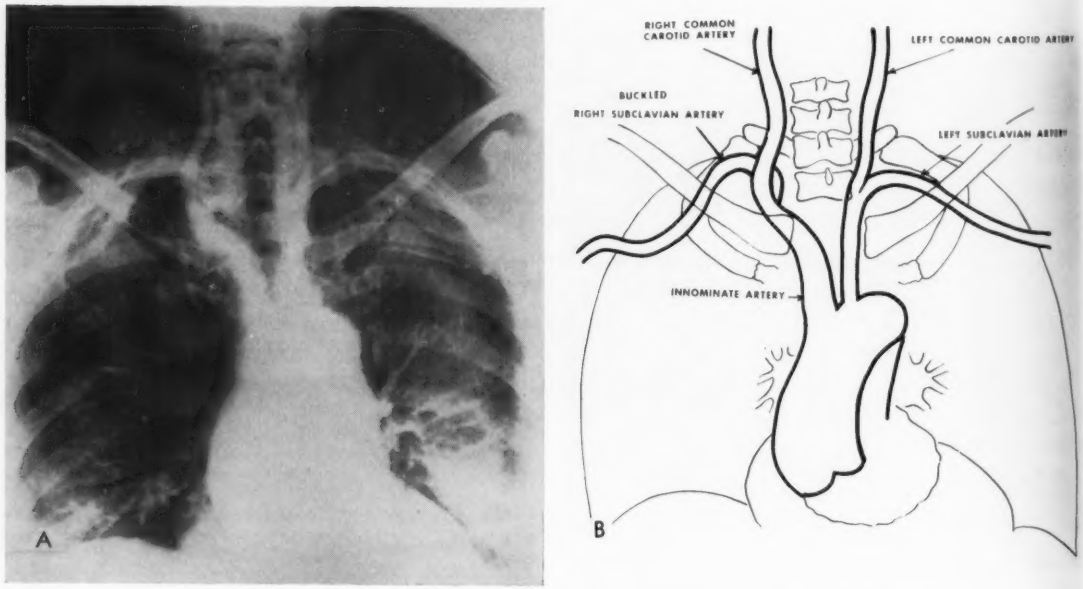

FIG. 16. Buckling of innominate and right subclavian arteries. (*A*) Frontal angiocardiogram. (*B*) Tracing of *A*.

subclavian artery aneurysms in a study of 716 cervical rib cases. Case reports by Lindskog and Howes,[39] MacFee,[43] Eden,[16] Graham,[25] Imler and co-workers,[34] Steinberg,[74] and Poppen and co-workers[58] total 44 cases. Older studies of cervical rib cases record no aneurysmal complications.[1,77] Subclavian artery aneurysms associated with cervical ribs always occur in the third portion of the subclavian artery, distal to the site of impingement of the rib on the vessel; the dilatation is poststenotic. The mechanism of poststenotic dilatation of the subclavian artery was first studied by Halsted and Reid[26] who reproduced poststenotic dilatation in the abdominal aorta of dogs. More recently, Holman[29] has investigated the problem. Doubt has arisen concerning the aneurysmal nature of such lesions since resolution of the vascular abnormality occurs when the obstructing element is removed. The symptoms associated with subclavian artery aneurysms are swelling of the upper extremity, numbness, pain, and gangrene of the fingertips. Physical examination may reveal a pulsatile mass and bruit in the supraclavicular area with diminution and obliteration of the pulse and blood pressure. Angiocardiography establishes the diagnosis (Fig. 17).[74] Subclavian artery aneurysms due to cervical ribs, in contrast to anteriosclerotic aneurysms, do not tend to rupture. In the earlier literature, surgery for subclavian artery aneurysms was not advised; simple application of supraclavicular pressure was thought to be sufficient to reverse complaints. More recently, however, a more radical approach, resection of the cervical rib or scalenus anticus muscle, has been advocated and found to be curative.[74]

(2) Syphilitic Subclavian Artery Aneurysms. Syphilis is not a common cause of aneurysms of the subclavian artery. Kampmeir[35] reviewed 1,163 patients with syphilitic aneurysms and found only 2 cases involving the subclavian artery. A review of the literature yielded 4 additional cases.[12,18,76,85] Syphilitic subclavian artery aneurysms are usually large and encroach upon the supraclavicular and intrathoracic spaces. Rib erosion and destruction are common; rupture and fatal hemorrhage may also occur. Unilateral clubbing of the fingers and palmar contractures resembling Dupuytren's contracture have also been described. Swelling of the neck and a mediastinal mass may at first suggest a

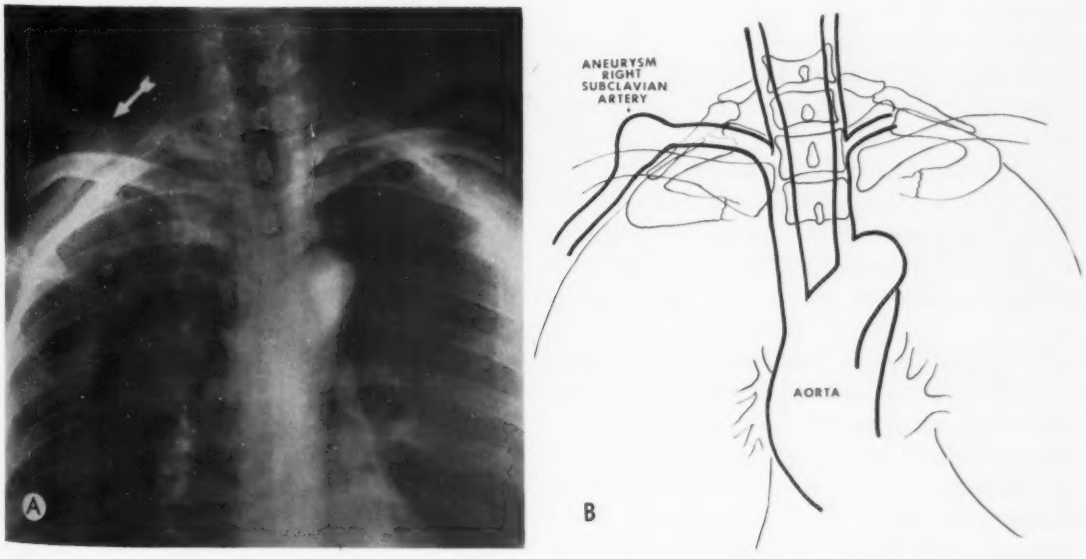

Fig. 17. Aneurysm of right subclavian artery due to cervical rib. (*A*) Frontal angiocardiogram. (*B*) Tracing of *A*. By permission of *The New England Journal of Medicine*.[74]

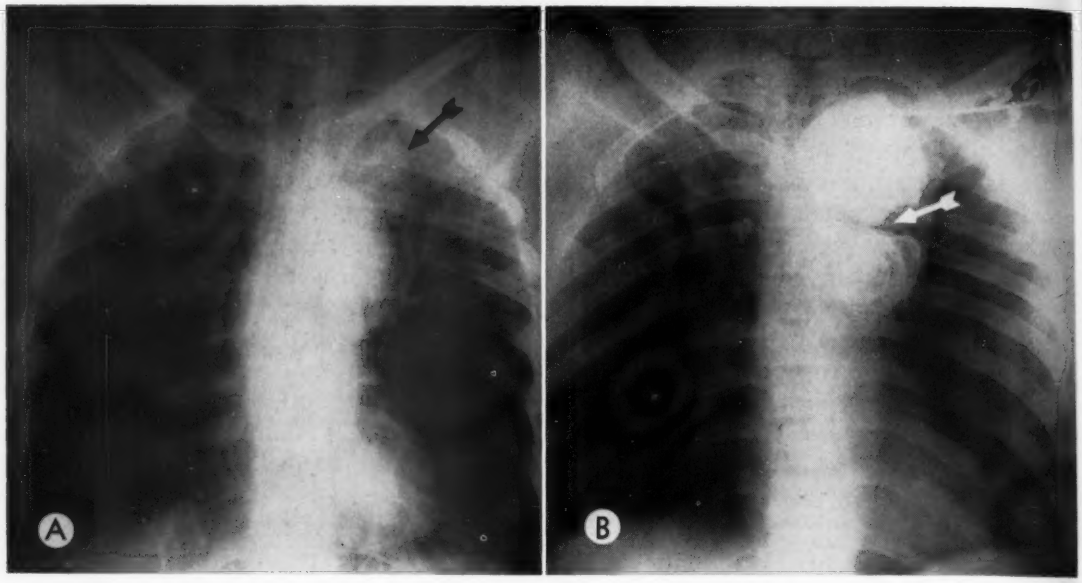

FIG. 18. Aneurysm of left subclavian artery due to syphilis. (*A*) Frontal angiocardiogram. Arrow points to poorly opacified aneurysm. (*B*) Arteriogram showing opacified left subclavian artery aneurysm. Note narrow neck of aneurysm (arrow) which prevented adequate opacification with angiocardiographic technique. The aortic arch has been wired. By permission of *The British Journal of Radiology*.[76]

nonvascular tumor but clinical and angiographic studies establish the diagnosis (Fig. 18).

(3) Arteriosclerotic Subclavian Artery Aneurysms. Arteriosclerotic aneurysms of the brachiocephalic vessels may be expected to become more prevalent owing to increasing longevity. Early diagnosis is important because resection of the aneurysm and replacement by homologous grafts will prevent rupture. Arteriosclerotic aneurysms should be suspected in patients with generalized arteriosclerosis who have superior mediastinal masses and diminution of the ipsilateral pulse. Calcification is often seen within the mass at the origin of the subclavian artery or along its course (Fig. 19).[61,73]

(4) Traumatic Subclavian Artery Aneurysms. Traumatic aneurysms of the subclavian arteries are chiefly due to penetrating wounds of the chest and case reports are abundant in the military literature.[17,68] Many are due to fractures of contiguous osseous structures, chiefly the clavicle. Comparatively minor trauma may give rise to aneurysms. When the aneurysm is located in the first part of the subclavian artery, there is no palpable tumor of the neck, since it is completely intrathoracic. Such cases may be accompanied by Horner's syndrome. The treatment is surgical and accurate preoperative localization is important because it determines the approach. For instance, the transpleural route

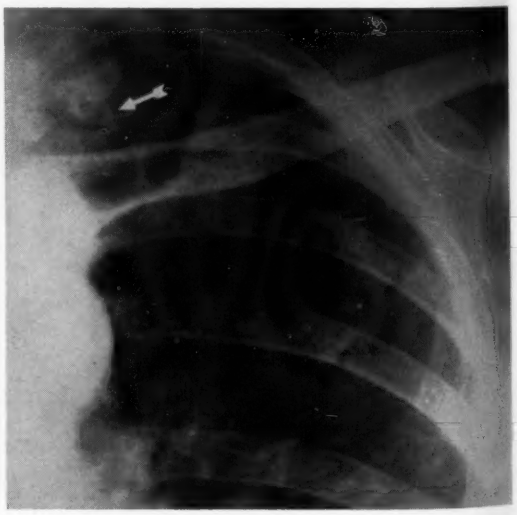

FIG. 19. Arteriosclerotic aneurysm of left subclavian artery. Note calcification of aneurysm (arrow).

is essential for treatment of aneurysms of the first part of the subclavian artery.[80]

Aneurysmal involvement of the left subclavian artery in traumatic aneurysms of the thoracic aorta has been noted by Sarot.[64] The mechanism of aortic rupture with development of a chronic traumatic aneurysm at the top of the aorta just distal to the origin of the left subclavian artery has been recently reported.[72]

d. Subclavian Arteriovenous Fistulas.

Arteriovenous fistulas of the subclavian artery are of two types, the congenital and acquired. Both produce abnormal shunting of blood with consequent serious alterations of circulatory dynamics. Arteriovenous fistulas tend to enlarge and eventually cause cardiac enlargement, arrhythmia and congestive failure. Locally, there is distention of the afferent artery with degenerative changes in its wall. There is also dilatation of the vein with thickening of the wall, while extensive collateral circulation develops distal to the fistula. Clinically, there is a continuous bruit, accentuated on systole, and hypertrophy of the affected part. Arteriovenous fistulas of the subclavian artery (Fig. 20) are chiefly traumatic in origin resulting from penetrating wounds[21] such as those produced by shell fragments in the supraclavicular area. Loud continuous bruits, decreased radial pulses, increased venous pressure and circulation times are characteristic. Arterial repair rather than ligation is advised because ligation produces ischemia on exercise of the extremity. Subclavian arteriography will localize the lesion prior to treatment and show the continuity of the vessel following repair.

The occurrence of endarteritis at the site of an acquired arteriovenous fistula is uncommon; only 15 patients with this complication are reported in the American literature. In a recent report, Hook *et al.*[32] described 2 such patients. In one, the left subclavian artery was the site of the fistula. Cardiovascular studies revealed decreased systolic and diastolic blood pressures of the involved side, increased stroke volume, increased blood volume and slowing of the pulse on compression of the fistula. The heart was enlarged and pulmonary congestion was present. Repair of the fistula was delayed for three years until the patient became febrile. The left arm was then larger than the right and there was extensive collateral circulation about the shoulder. Roentgenography showed a large, irregular, calcified mass in the region of the aneurysm. Despite antibiotic drugs the patient did not improve. Following surgical excision of the arteriovenous fistula, however, the fever gradually abated. Culture of the specimen revealed *Micrococcus pyogenes* var. *albus*.

Fig. 20. Angiocardiogram showing traumatic arteriovenous fistula of left subclavian artery.

Experimentally in dogs, Lillehei and coworkers[38] have produced bacterial endarteritis and endocarditis by construction of large arteriovenous fistulas. Endarteritis at the site of the fistula was frequent after long periods of observation.

e. Occlusion of the Subclavian Arteries.

(1) The Subclavian Arteries in Aortic Arch Syndrome (Pulseless Disease). Absence of radial pulses was described in medical reports as early as 1839,[63] and was undoubtedly known to physicians even before this. Postmortem examination demonstrated occlusion of the great vessels

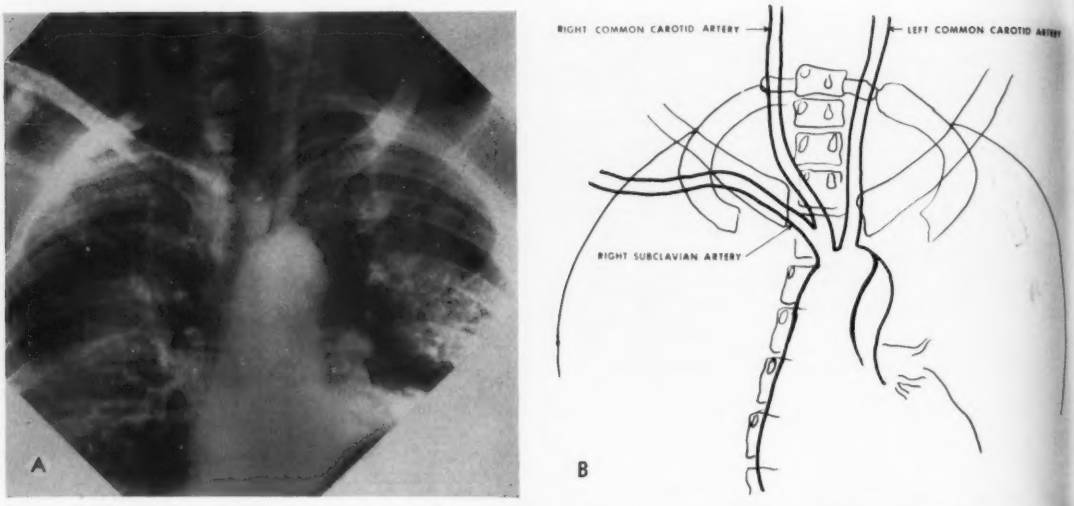

FIG. 21. Syphilitic thrombosis of the left subclavian artery. (*A*) Frontal angiocardiogram. (*B*) Tracing of *A*. Note absent left subclavian artery opacification.

arising from the aortic arch. Among the chief causes of aortic arch syndrome are syphilis (Fig. 21), arteriosclerosis and arteritis (Fig. 22 and 23). Rarer etiologies are obstruction due to tumors and congenital anomalies. It was not until 1908, however, that another form was described by the Japanese ophthalmologist, Takayasu.[78] In 1952 similar reports began to appear in the American literature.[7,8,24,83] The etiology of this disease is attributed to chronic progressive inflammation (arteritis) involving the brachiocephalic vessels leading ultimately to blindness and death. This syndrome has been termed "obliterative brachiocephalic arteritis," "pulseless disease," "reversed coarctation," and "Takayasu's disease."

Takayasu's disease (Fig. 22 and 23) should be suspected when transitory or progressive blindness, cataracts, atrophy of the iris, peripapillary venous anasto-

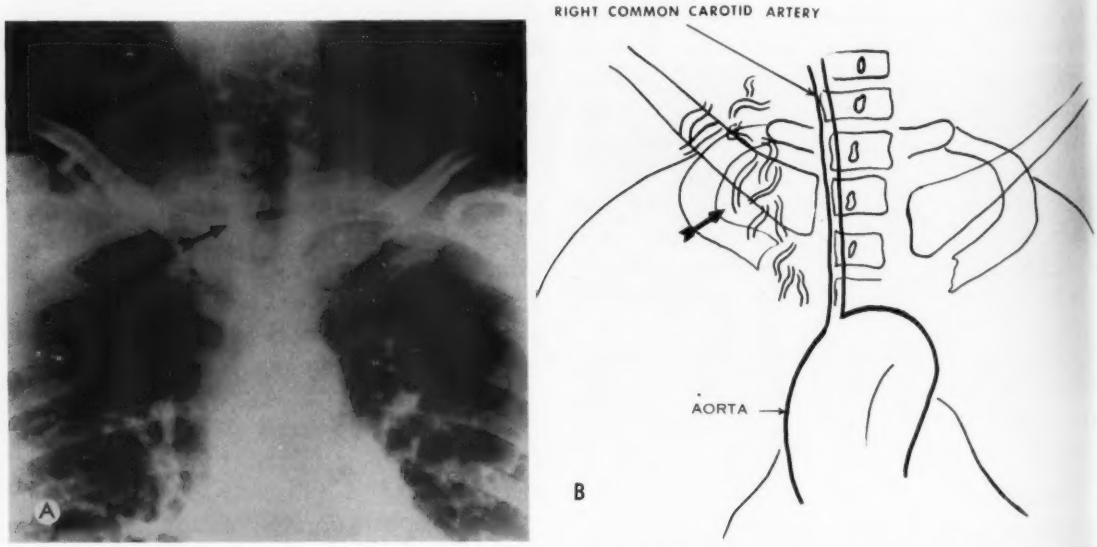

FIG. 22. Pulseless disease of many years' duration in a thirty-eight year old woman with multiple lipomatosis. (*A*) Frontal angiocardiogram shows only opacification of the right common carotid artery (arrow). (*B*) Tracing of *A*. Arrow points to abundant collaterals.

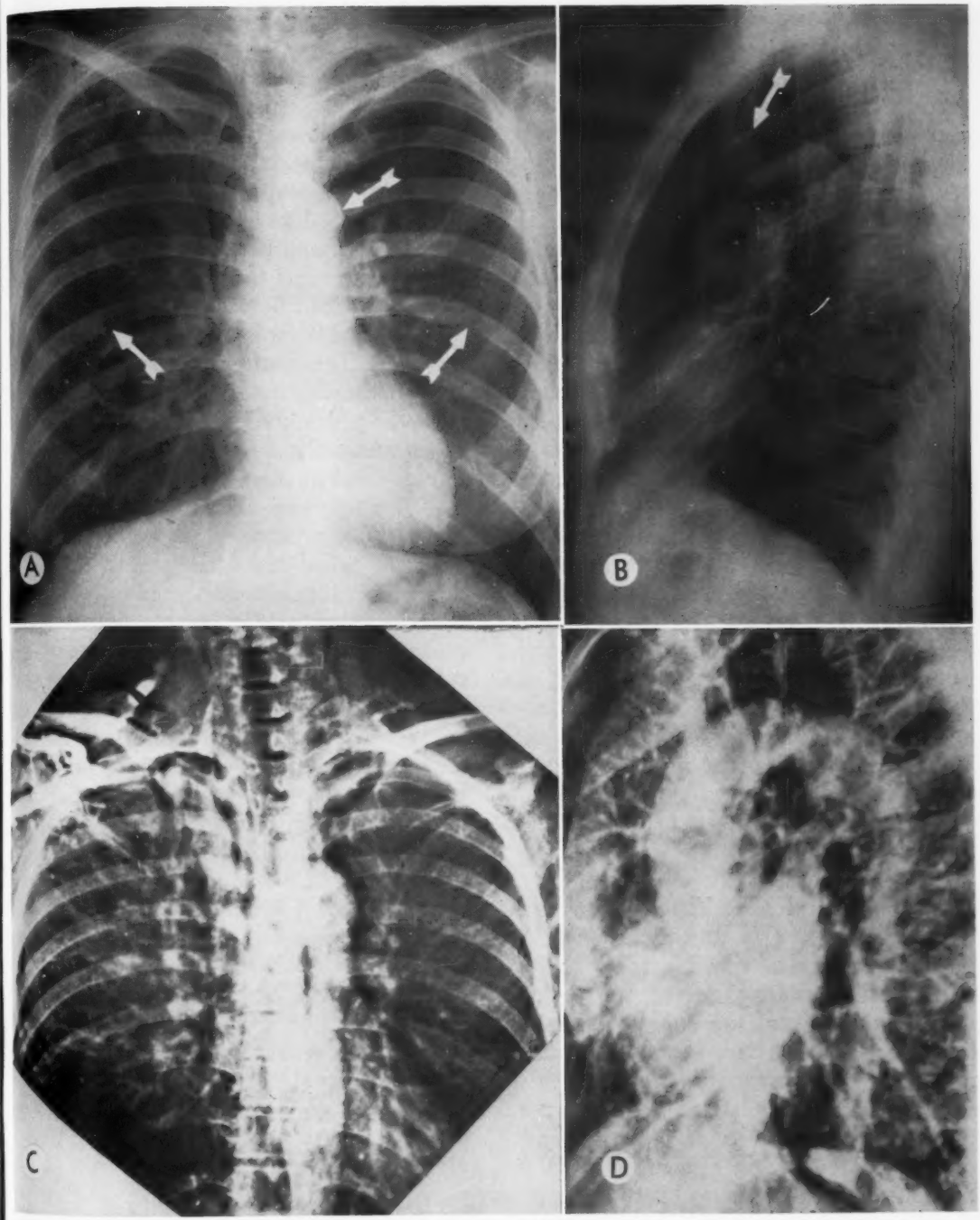

FIG. 23. Takayasu's disease in a thirty-five year old Japanese woman with absent pulses and complaints of syncope, headaches and episodes of blindness. (*A*) Frontal roentgenogram shows calcification of aortic arch (arrow) and notching of ribs (arrows). (*B*) Lateral roentgenogram more clearly reveals the aortic arch calcifications (arrow). (*C*) Frontal (LogEtronic) angiocardiogram shows only the right carotid artery to be patent. (*D*) Left oblique (LogEtronic) angiocardiogram again shows only the right common carotid artery. (Courtesy of Dr. William Dubilier, Jr.[45])

moses, atrophy of the facial muscles, perforation of the nasal septum, claudication of the masticator muscles, syncope, seizures and absent radial pulses occur in young women. Laboratory studies are not significant except for elevation of the erythrocyte sedimentation rate. This has been interpreted as evidence of arteritis. The chest roentgenogram may show rib notchings if extensive collateral circulation develops (Fig. 23*A*). The hands are free of trophic changes because the collateral circulation is abundant and adequate. The mean arterial pressure of the extremities is maintained but oscillometric studies reveal dampened or absent pulsations. Other diseases such as severe aortic stenosis, thoracic inlet syndrome and superior mediastinal tumors simulate pulseless disease and must be differentiated. Therapy with steroid hormones, anticoagulants and penicillin have been advocated and tried without arresting the disease. However, there is a recent report of dramatic improvement in arteriosclerotic aortic arch involvement following long term anticoagulants.[69] In another recent report improvement following combined steroid and anticoagulant therapy has been described.[36A]

(2) Thrombosis of the Subclavian Artery Due to Cervical Rib. Thrombosis of the subclavian artery is an infrequent complication of cervical ribs. However, reviews indicate that about 50 per cent of patients with thrombosis of the subclavian artery due to cervical ribs have severe sequelae. Of a total of 33 cases recently reported,[65,67] 11 suffered loss of a phalanx or digit, 2 required upper extremity amputation, and 3 developed hemiplegia due to proximal extension of the thrombus. It would appear that patients with cervical ribs and vascular symptoms in the upper extremity require careful study to decide if operation is indicated.

Schein, Haimovici and Young[65] reported 2 cases and reviewed 29 cases found in the literature. The ages ranged from sixteen to sixty-six years; 73 per cent of the patients were between twenty-one and fifty years. The symptoms were pain, pallor, paresthesias, coldness, decreased exercise tolerance, weakness and trophic changes. The onset of thrombosis was often due to relatively minor trauma such as lifting, stretching or carrying. A supraclavicular mass was palpable in all patients due to the thrombosed vessel and periarterial inflammation. Sixty per cent of the cases were males and 60 per cent had the lesion on the right side. The cervical rib was complete in all but one instance. The authors stressed that even though the thrombosis was at the level of the subclavian artery, there was often disease proximally and distally leading to extension and involvement of the common carotid artery and contralateral hemiplegia. Peripheral occlusions of the axillary, brachial, radial and ulnar vessels may also occur due to embolus. Especially dangerous is proximal extension of the thrombus because of compromise of the collateral circulation between the first part of the subclavian artery and the first and second parts of the axillary artery. For these reasons, arteriography is advised for preoperative evaluation of the arterial tree of the involved upper extremity and for exact localization of the lesion.[65]

Shenkin[67] reported 3 new cases of subclavian artery thrombosis, all on the right side with complete cervical ribs. Duration of symptoms prior to thrombosis was three, seven and twelve years. The patients had been previously examined and the cervical rib recognized but conservative management was prescribed. The author, however, advocates surgery despite mild symptoms if there is arterial compression by a cervical rib. The elective operation is decompression of the subclavian artery which is a relatively simple procedure, although, depending upon the anatomic findings, cervical costectomy or interruption of the sympathetic nerve supply by stellate ganglion block may be necessary. Arterial grafting, thrombectomy, arteriectomy and dorsal sympathectomy have also been found to produce improvement. Prophylactic

decompression of the contralateral subclavian artery is not indicated, since bilateral thrombosis has not been reported.

(3) Thrombosis of the Subclavian Artery Due to Trauma. Thrombosis of the subclavian artery secondary to regional trauma, while not rare, is infrequently reported because of the obvious pathogenesis (Fig. 24).

f. Involvement of the Subclavian Arteries by Tumors.

Tumors in the region of the lung apices have special features which distinguish them from those in other areas of the thorax. The earliest report of an apical tumor was made in 1838 by Hare.[27] He described a forty year old male with a rapidly growing tumor of "the inferior triangular space" in the left side of the neck. The initial complaint was shoulder pain with numbness and tingling along the ulnar nerve distribution. A few months later, contraction of the left pupil developed, and the "elevator palpebrae ceased to perform its office." The patient died after four months and, at autopsy, a hard, nodular mass involving the arteries, veins and brachial plexus was found. A tumor extended under the clavicle and surrounded the subclavian artery and vein destroying the anterior scalene muscle. Following Hare's original report, MacDonnell[42] described a case with obliteration of the third portion of the subclavian artery. Ricaldoni in 1918 and Freeman[22] in 1921 added additional cases involving the subclavian artery.

In 1924, Pancoast[51] independently described 3 patients who complained of shoulder pain because of tumors arising at the superior thoracic inlet. On examination there was an apical tumor, destruction of ribs and vertebrae, lid lag, miosis of the pupil and absence of sweating of the affected side. Histologically, the tumors were squamous cell carcinomas and produced symptoms by invasion of the brachial plexus, cervical sympathetic chain and adjacent osseous structures. He believed that the tumors originated from embryonal tissue of branchial rests. Other papers described the extrapulmonary origin of

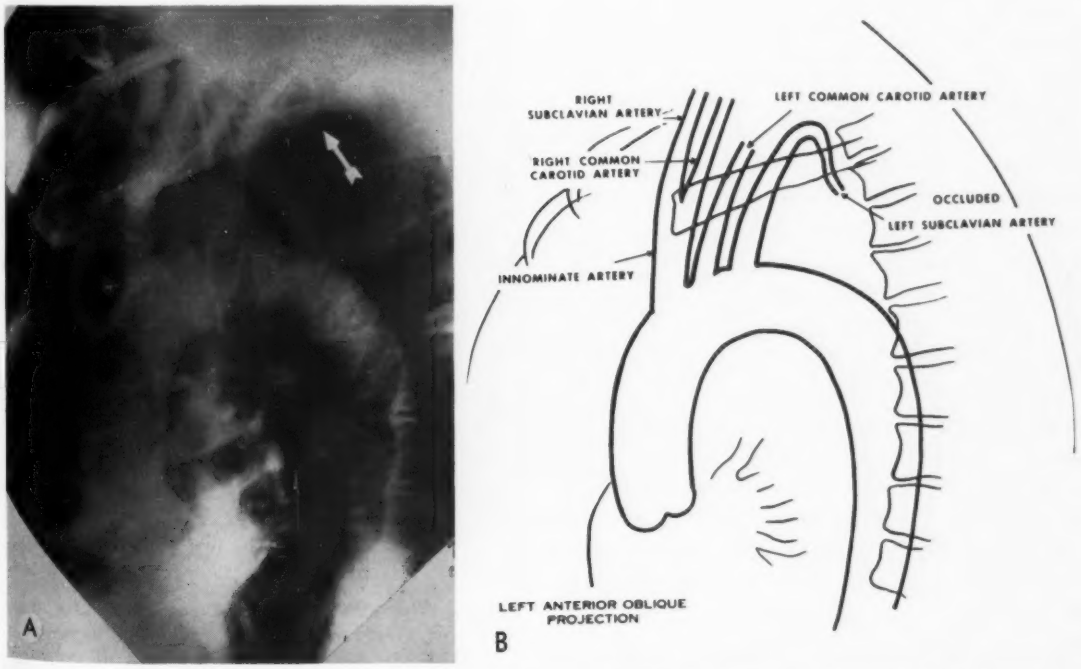

Fig. 24. Thromobosis of the left subclavian artery secondary to fractured clavicle. (*A*) Left oblique angiocardiogram showing the occluded left subclavian artery. (*B*) Tracing of *A*.

Fig. 25. Angiocardiogram revealing involvement of left subclavian artery by left apical bronchogenic carcinoma (Pancoast syndrome).

such tumors and suggested the term "branchioma."[23] The true nature of such tumors still remains incompletely understood. Conservative pathologists insist that serial sections of adjacent lung tissue must be thoroughly studied before an extrapulmonary origin can be established. Very often, inadequate reports state that the tumor is easily separable and extrapleural, or that bronchial and lung tissue structures are absent. It is probably more correct to name tumors in this region tumors of the thoracic inlet producing the Pancoast syndrome rather than calling them Pancoast tumors.[6] Many lesions at the apex of the lung may produce the Pancoast syndrome because the symptoms produced by apical masses are due to the involvement of the anatomic structures in the area.

Subclavian artery involvement by apical tumors was first reported by Hare[27] and confirmed by MacDonnell.[42] Browder and De Veer[6] also described disturbances in blood pressures and pulses with apical tumors. Herbut and Watson,[28] in an extensive review of the literature, found 151 cases of apical tumors and noted edema of the arm indicating vascular involvement in 20 instances. Chardack and MacCallum[10,11] report an unusual case with five year survival. Our experience with 6 patients with the Pancoast syndrome showed 5 to have involvement of the subclavian artery or vein. The immediate cause of death in one case was erosion of the subclavian artery (Fig. 25).

III. Miscellaneous Involvement of the Subclavian Arteries

Abnormalities of the subclavian artery occur in many other conditions which will be discussed as a miscellaneous group.

a. The Subclavian Artery in Interruption of the Aortic Arch.

Interruption of the aortic arch (Fig. 26)[44] is a rare congenital anomaly consisting of absence of the aortic arch between the origin of the left subclavian artery and the point of entrance of the ductus arteriosus, a segment known as the isthmus. Absence of the isthmus is an extreme form of "juvenile" coarctation. The ascending aorta

Fig. 26. Absence of left subclavian artery due to interruption of aortic arch. The great vessels are transposed; the aorta arises from the right ventricle. (From St. Francis Hospital and Sanatorium, Roslyn, New York.)

supplies the head and upper extremities with blood from the left ventricle causing them to be pink. The descending aorta supplies the lower extremities with unsaturated blood from the pulmonary artery causing cyanosis of the legs. Occasionally, the left subclavian artery arises from the descending aorta and the left upper extremity is cyanotic. Merrill *et al.*[46] discuss the embryonic development of the left subclavian artery and believe that its origin from the descending aorta represents involution of the isthmus at an earlier stage. They report the first case of successful repair of interruption of the aortic arch.

b. Subclavian Artery Occlusion following Blalock-Taussig Operation.

In the Blalock-Taussig operation (Fig. 27) the proximal portion of the subclavian artery is anastomosed to the pulmonary artery. There are usually no serious sequelae because rich collaterals prevent gangrene of the upper extremity. However, the existence of abnormal circulation prior to operation may cause complications. Taussig[79] reported gangrene in a patient with preoperative absence of the radial pulse and hemiatrophy of the trunk. Webb and Burford[82] added an additional case and thought that spontaneous thrombosis of the collateral circulation was probably due to polycythemia.

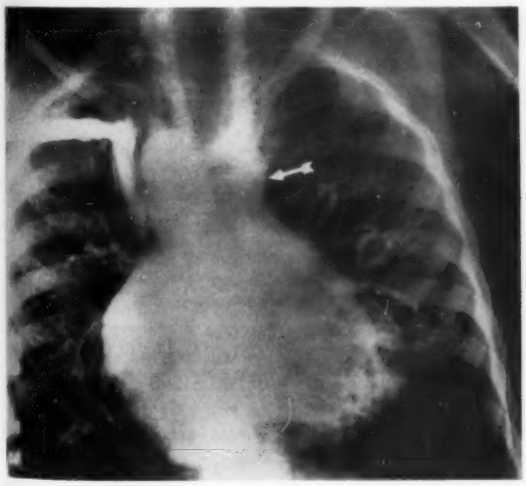

Fig. 27. Angiocardiogram showing the left subclavian artery anastomosed to left pulmonary artery (Blalock-Taussig operation). Arrow points to site of artificial ductus.

Fig. 28. Angiocardiogram showing deformity of left subclavian artery (arrow) following thoracoplasty for pulmonary tuberculosis.

Following the Blalock-Taussig procedure, the subclavian-pulmonary artery anastomosis may become occluded. Levin and Rigler[37] and Peterson[57] report many instances of unilateral rib notching following occlusion of the shunt. The average time interval between occlusion and the roentgenographic demonstration of rib notching was twenty-three months, but in one case, it was only six months.

Findlay and Maier[20] reviewing systemic anomalies of the pulmonary circulation, describe 5 patients with left subclavian artery communications with the left pulmonary artery. These all had associated anomalies; right sided aorta, pulmonary atresia, patent interventricular septum and pulmonic stenosis. In 2 cases the right subclavian artery communicated with the hilus and was associated with aortic stenosis, right pulmonary artery atresia and a small bilobed right lung. Therefore, in planning surgery, thorough preoperative studies are desirable to forestall hemorrhage from injury of anomalous vessels.

FIG. 29. Angiocardiogram showing marked angulation of left subclavian artery associated with kyphoscoliosis. By permission of *Radiology*.[15]

c. Subclavian Artery Deformity Resulting from Thoracoplasty and Kyphoscoliosis.

A case with marked distortion and angulation of the left subclavian artery following thoracoplasty was associated with a vascular bruit (Fig. 28). Another patient with severe kyphoscoliosis had pronounced distortion and elongation of the subclavian arteries. Such a case is shown in Figure 29.[15]

SUMMARY AND CONCLUSIONS

Angiocardiography, by visualizing the cardiovascular system, made possible the roentgen study of the subclavian arteries. Utilizing the angiocardiographic experience of a twenty year period, normal standards for length and caliber of the subclavian arteries are established. Alterations in disease, congenital and acquired, are also described and classified. Finally, the academic and practical significance of data concerning the subclavian artery in health and disease is discussed.

Israel Steinberg, M.D.
The New York Hospital
525 East 68th Street
New York 21, New York

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THE NORMAL AXILLARY VENOGRAM*

By C. JULES ROMINGER, M.D.
PHILADELPHIA, PENNSYLVANIA

DIVERGENT views have been expressed in the literature regarding the interpretation of certain venographic findings in the subclavian and axillary veins in cases of suspected axillary vein thrombosis.[1,2,3,5,15,16,17] Erroneous interpretations have been made in the past by trying to correlate a dynamic roentgenographic picture with static anatomic descriptions.

The lack of a well documented series of axillary venograms a few years ago was understandable, since contrast media had not been developed with the safety of present media.[1,2,4,12,16,17] Hundreds of angiocardiograms have been performed in recent years utilizing these arm veins, but the authors have focused their attention on descriptions of the endothoracic vascular structures.[8] In contrast to an adequate consideration of the leg veins, most radiology texts briefly dismiss the subject of the arm veins.[3,5,6,7,8,9,14] They present a classic anatomic description of the arm veins without any consideration of the vascular dynamics. The only published study which fully considers the dynamics of the axillary venogram is that of Tagariello[15] analyzing 32 cases. No studies of the normal mensuration of the axillary vein are available.

It is the purpose of this paper to describe the normal axillary venogram in the adult.

METHOD AND MATERIAL

The normal arm veins of 100 individuals were examined by making a roentgenogram of the arm and shoulder area during the injection of a contrast medium for routine intravenous urography. The medium used in all patients in this study was 76 per cent renografin.* This medium was injected in 20 cc. doses for intravenous urography. The first 10 cc. was injected within three seconds through an 18 gauge needle. During this portion of the injection, a roentgenogram of the shoulder was made using non-Bucky technique, and with the patient maintaining normal quiet respiration. The remainder of the medium was then injected at a slower rate. No side effects attributable to the contrast medium were noted in this series of patients.

Fifty left axillary veins and 50 right axillary veins were examined. The distribution of patients between the two sexes was equal.

The age distribution of the patients was as follows:

Age	*Male*	*Female*
20–30	7	9
31–40	5	6
41–50	9	7
51–60	14	14
61–70	8	7
71–80	6	7
80–	1	0

SITE OF INJECTION

The site of injection was found to have an influence on the veins visualized, as would be expected from classic anatomic descriptions of these veins (Table 1).

Early in the course of this study it was noted that injection into the cephalic vein did not often give a good visualization of the axillary vein (Fig. 1). In 8 cephalic vein injections the axillary vein was opacified in only 3 instances. The cephalic vein itself was visualized in each instance, the basilic vein in 5, and the brachial vein in 2. The visualization of the basilic and brachial veins was accomplished by passage of the medium through communicating veins, but the axillary opacification usually resulted from retrograde flow from the cephalic

* Renografin ® Squibb sodium and methylglucamine diatrizoates.

* From Misericordia Hospital, Philadelphia, Pennsylvania. Presented in exhibit form at the Forty-second Annual Meeting of the Pennsylvania Radiological Society, Williamsport, Pennsylvania, May 17–18, 1957.

TABLE I

FREQUENCY OF VISUALIZATION OF VEINS AS RELATED TO SITE OF INJECTION

Veins Visualized	Antecubital Vein Injection 48 Persons	Basilic Vein Injection 44 Persons	Cephalic Vein Injection 8 Persons
Axillary Vein	42	36	3
Cephalic Vein	45	7	8
Basilic Vein	48	44	5
Brachial Vein	16	2	2
"Brachial Plexus" of Veins	14	1	0

axillary junction to the valve at the basilic-axillary junction (Fig. 2).

The antecubital vein and the basilic vein were both found to be satisfactory routes of injection for an axillary venogram. It was sometimes difficult to distinguish between these two veins in the antecubital fossa. More of the arm veins were visualized after antecubital injection than basilic injection. As was expected from the anatomic descriptions, both the basilic and cephalic system were seen in practically every instance following antecubital injection (Fig. 3).

Our data show a slightly lesser percentage of good axillary vein opacification following basilic vein injection as compared to antecubital vein injection. This was due to

FIG. 2. Injection of cephalic vein with retrograde filling of axillary vein. This occurs infrequently with cephalic vein injection. Male.

the "streaming effect," a commonly noted effect in angiography (Fig. 4). This layering of the denser contrast medium was seen in 16 cases of this series, 10 basilic vein injections and 6 antecubital vein injections. It appeared to be related to timing of the roentgenographic exposure, as well as to the viscosity of the medium. It is my impression that it was more often seen at the "leading edge" of the contrast bolus, and that if the roentgenographic exposure were delayed until at least five to ten seconds after starting the injection, it could be completely avoided.

Antecubital vein injection in 48 persons

FIG. 1. Injection into cephalic vein usually demonstrates only this vein and a good axillary venogram is generally not obtained. Female.

FIG. 3. Antecubital vein injection demonstrating a trifid basilic vein with good visualization of all other major arm veins. Female.

Fig. 4. Antecubital vein injection showing a marked streaming effect in axillary vein. Male.

Fig. 5. Average male axillary vein. Basilic vein injection. Note tapered proximal end of vein due to partially closed cusps.

resulted in good opacification of the axillary vein in 42, the cephalic vein in 45, basilic vein in 48 and brachial vein in 16.

Basilic vein injection in 44 persons resulted in good opacification of the axillary vein in 36, the cephalic vein in only 7, the basilic vein in all instances, and the brachial vein in only 2 instances.

Gross Character of Veins

There was no demonstrable difference in the venograms of the right and left arm. The age of the subject had no influence on the gross character of the veins, their measurements, or valvular characteristics.

There were striking differences in the veins of males and females. In general, the male arm veins (Fig. 5) are straighter and of greater diameter than those of the female (Fig. 6). They have fewer valves and less prominent paravalvular sinuses. Fewer small tributaries are seen, and there are fewer anatomic variations.

The female arm veins are more undulating and smaller in diameter than those of the male (Fig. 6). Their valves are more prominent and numerous. More tributaries and anatomic variations are visualized.

Roentgenograms of the arm veins of most persons of both sexes presented the standard anatomic textbook pattern. The antecubital fossa usually contains three veins. One of these is the cephalic, which continues up in the lateral biceps furrow and runs in an arch above the shoulder joint, emptying into the axillary vein after its junction with the thoraco-acromial vein. The basilic vein runs in the medial biceps furrow and at about the junction of the lower and middle one-third of the paired brachial veins which usually follow the artery. This junction forms the axillary vein and usually occurs at the lower border of the tendon of the teres major muscle. The third vein normally found in the antecubital fossa is the antecubital vein

Fig. 6. Average female basilic vein injection with partial retrograde filling of cephalic vein and filling of several small tributaries of axillary vein. Proximal end of vein is tapered due to partially closed cusps.

FIG. 7. Antecubital vein injection. Note filling of all major veins and a large group of communicating veins between the cephalic and basilic systems which we have chosen to call the brachial plexus of veins. Female.

itself, which communicates with both of these basic systems.

On roentgenograms we consider the axillary vein as beginning at the lateral scapular border and becoming the subclavian vein at the lower border of the first rib. The subclavian vein is normally visualized with the technique used in this series only to its junction with the internal jugular vein. Here the medium becomes diluted. The innominate vein, superior vena cava and heart are not visualized as they would be if a large bolus of contrast medium more rapidly injected were used, and serial exposures made as for angiocardiography.

Fifteen patients presented a striking group of parallel veins coursing obliquely cephalad from the brachial vein to the basilic vein (Fig. 3 and 7). This group of veins I have chosen to call the brachial plexus of veins, since as far as I am aware, they have no special name. They possibly occur with greater frequency than indicated by this figure. Their opacification appears to be related to the site of injection, since they were seen in 14 of the 48 antecubital injections, in 1 of the 48 basilic vein injections, and in none of the cephalic vein injections.

The basilic vein was visualized as bifid in

TABLE II

GROSS CHARACTER OF VEINS

	Male 50 Persons	Female 50 Persons
Bifid Basilic Vein	12	5
Brachial and Basilic Junction at Second Rib	1	3
Loop in Basilic Vein	1	1
Cephalic Vein United with Brachial Vein	—	3
Trifid Basilic Vein	—	1
Loop in Cephalic Vein	—	1
Racemose Cephalic Vein	—	1

12 males and in 5 females (Table II). In female it was trifid. A loop was seen in the basilic vein in 1 male and 1 female (Fig. 8).

The brachial and basilic vein did not join until they had reached the level of the second rib (average junction at inferior scapular border) in 3 females. In 1 female the cephalic vein contained a loop and in another it was quite racemose (Fig. 9).

MEASUREMENT OF THE VEINS

The measurements of the various veins were made directly on the roentgenograms to the nearest millimeter (Table III). No attempt was made to correct for distortion (the target film distance was 36 inches in each instance) or to try to judge the degree of spasticity, if any, produced by the in-

FIG. 8. Basilic vein injection. Note loop in basilic vein and lack of filling of cephalic vein. Female.

TABLE III

DIMENSIONS OF VEINS
(in millimeters)

Vein	Male		Female	
	Average	Range	Average	Range
Axillary Vein at Scapula	10	(8–16)	8	(6–16)
Axillary Vein at First Rib	13	(8–19)	11	(7–14)
Axillary Vein at Valve	15	(9–21)	14	(7–18)
Cephalic Vein	8	(5–11)	7	(4–10)
Cephalic Vein at Valve	10	(6–13)	8	(6–10)
Basilic Vein at Valve	11	(7–15)	10	(5–18)
Small Tributaries at Valve	6	(5– 7)	5	(3– 7)

jection. These two factors were felt to be very slight, and to compensate for one another. No correlation was found between the diameter of a subject's arm and the vein size.

In the males the average diameter of the axillary vein at the lateral scapular border was 10 mm. (8–16 mm. range), at the first rib 13 mm. (8–19 mm. range) and at the largest valve 15 mm. (9–21 mm. range). In the females the average diameter of the axillary vein at the lateral scapular border was 8 mm. (6–16 mm. range), at the first rib 11 mm. (7–14 mm. range) and at the largest valve 14 mm. (7–18 mm. range). The average diameter of the cephalic vein in the males was 8 mm. (5–11 mm. range) and in the females 7 mm. (4–10 mm. range). The cephalic vein at its largest valve measured on the average 10 mm. in the males (6–13 mm. range) and 8 mm. in the females (6–10 mm. range). The basilic vein at its largest valve measured an average of 11 mm. in the males (7–15 mm. range) and of 10 mm. in the females (5–18 mm. range). The smaller tributaries of the axillary vein averaged 6 mm. in diameter in the males and 5 mm. in diameter in the females.

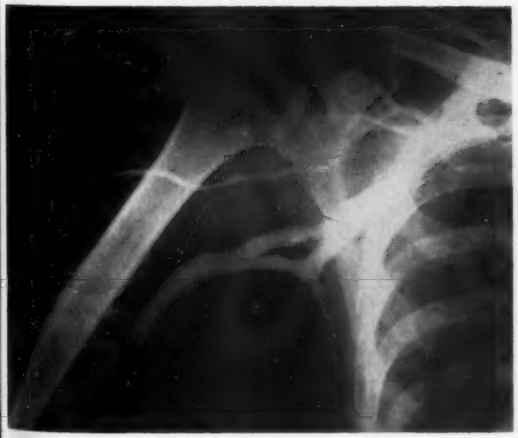

FIG. 9. Antecubital vein injection. Note bifid basilic vein and racemose cephalic system. Valves are demonstrated in several small tributary veins. Female.

CHARACTERISTICS OF THE VENOUS VALVES

One of the most striking features of the normal axillary venogram is the appearance of the valves (Table IV). These are visualized as bulges of varying degree in the venous outline, depending on the degree of distention of the paravalvular sinuses. The state of these valves has given rise in the past to confusion and misinterpretation of the images seen at venography as pointed out by Tagariello.[15] In some instances in the literature, futile operative procedures for nonexistent venous blockage have been carried out as a result of misinterpretation of the proximal subclavian vein valve image.

The results of this series of 100 venograms confirm the findings of Tagariello in his series of 32 venograms. I agree that the only valid roentgenographic evidence of subclavian vein obstruction is demonstrable *extensive collateral circulation* since the "images by blocking" and "stenosis"

TABLE IV

CHARACTER OF VALVES

		Male 50 Persons	Female 50 Persons
Number of Valves Visualized in Axillary Vein	1	16	20
	2	15	12
	3	4	2
	4	1	0
Depth of Cusps of Axillary Valves		7 mm. (6–11)	9 mm. (5–11)
Configuration of Proximal Valve (100 persons)			
Not Seen		22	
"Image by Blocking" Closed Valve		22	
"Image of Stenosis" Partial Closure		56	
Number of Basilic Vein Valves (97 persons)		3 (2–7)	3 (0–6)

described especially in the foreign literature are often found in the normal venogram. Since this collateral circulation is also visible on clinical examination, some authors have questioned the necessity of venography in establishing the diagnosis of axillary vein thrombosis.[3,5,10]

The venous valves consist of two thin cusps which vary in position. They lie adjacent to the venous wall when completely open and are not visible. As the valve closes, these two cusps move toward the midline of the vein and produce a progressive narrowing of the stream of contrast medium until they meet, completely closing the valve and stopping the flow of contrast medium. As the valve closes, the size of the paravalvular sinuses which are proximal to the cusps increases to a maximum at complete valve closure.[9] The saccular bulges thus produced are quite striking. They are a familiar sight to surgeons doing peripheral vascular surgery and are often noted during the axillary dissection of a radical mastectomy.

When axillary vein thrombosis or occlusion is suspected, these valvular images can and have given rise to confusing pictures that have, on occasion, been erroneously interpreted as an indication of blockage of the vein.

When the valve cusps are completely open, there is no suggestion of a block (22 per cent of this series). The vein is visualized as a straight tubular structure filled with contrast medium. Wave-like bulges are often seen at the valve sites due to slightly greater distensibility of the paravalvular sinuses (Fig. 10).

As the valve begins to close, the column of contrast medium is narrowed to a tapered conical shape, and the paravalvular sinuses expand (Fig. 5, 6 and 11). This is the venographic image that has been called the "image by stenosis" and misinterpreted as stenosis of the vein perhaps because of scarring or compression by the first rib, subclavian muscle, etc. This image is not pathologic but is a normal physiologic finding (56 per cent of this series).

When the valve closes completely, the column of contrast medium is shut off and its proximal end is concave (Fig. 12). The

FIG. 10. Antecubital vein injection. Basilic and cephalic systems seen. Note prominent paravalvular sinuses in the basilic system. Male.

Fig. 11. Effect of respiration on vein image. Injection of basilic vein showing bifid basilic system and brachial vein. Note very prominent paravalvular sinuses and closed valve in subclavian vein ("image by blocking") during the Valsalva maneuver. Female.

degree of concavity depends on the amount of back pressure on the valve. This is the so-called "image by blocking" and has been misconstrued to represent a thrombus in the vein when in reality it is a normal physiologic finding (22 per cent of this series).

In this series of venograms, the observations of Tagariello were confirmed. These "images by blocking and stenosis" were not

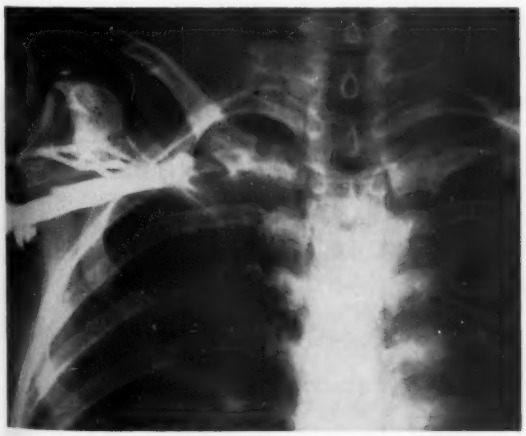

Fig. 12. Effect of respiration on vein image. Demonstration of closed valve in subclavian vein just distal to junction with jugular vein during the Valsalva maneuver ("image by blocking"). This is a normal finding and not evidence of obstruction. Female.

Fig. 13. Effect of respiration on vein image. Same patient as in Figure 11 and 12 demonstrating marked reduction in size of veins and paravalvular sinuses with arm adducted and injection made during quiet respiration.

related to any degree of abduction or adduction of the arm, nor to the position of the head and neck of the persons examined. Normal respiration did not influence their occurrence.

When the venogram was made with the subject inspiring against the closed glottis (Müller's maneuver), these valvular images were not seen (Fig. 13). Since Müller's maneuver decreases the endothoracic pressure and allows easier emptying of the veins into the vena cava, one would expect the valves to be completely open and therefore not visible. On the other hand, when the venogram was made with the patient executing forced expiration against the closed glottis (Valsalva's maneuver), the "image by blocking" was always seen (Fig. 12). The increase of intrathoracic pressure by this maneuver results in venous back pressure causing the valves to close and to be visible in varying degrees.

In serial study of several persons, the size of the paravalvular sinuses was also noted to change as increased venous pressure during the Valsalva maneuver caused them to become distended. Females had deeper paravalvular sinuses than males. The average depth of the sinuses in females was 9 mm. (range 5–11 mm.) and in males 7 mm. (range 6–11 mm.).

As indicated above, when the valves are completely open they are not visible in the venogram (30 per cent of this series). The number of valves visualized in any one venogram cannot be considered to represent the actual number present. However, in order to complete our description of the normal venogram, we counted the number of valves visualized in our subjects.

The number of valves seen in the axillary vein during normal quiet respiration varied. In 63 per cent of both males and females one or two valves were seen. Three valves were seen in 4 males and in 2 females and in 1 male four valves were demonstrated. An average of three valves was demonstrated in the basilic vein (range 2–7 male, 0–6 female). In all subjects only one valve was demonstrated in the cephalic vein.

SUMMARY

1. Axillary venography was performed on 100 normal persons.

2. The normal roentgenographic anatomy of the axillary vein and its tributaries is described. Their normal mensuration is presented.

3. The valvular images are the most striking feature of the venograms. On occasion in the past, these valvular images have been erroneously interpreted as evidence of venous thrombosis. The physiologic basis of these varied normal valvular images is discussed.

Misericordia Hospital
Department of Radiology
54th and Cedar Avenue
Philadelphia 43, Pennsylvania

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AZYGOS VEIN DILATATION SIMULATING MEDIASTINAL TUMOR*

By W. H. SHUFORD, M.D., *and* H. S. WEENS, M.D.
ATLANTA, GEORGIA

THE roentgenologic appearance of the azygos vein has been the subject of several studies.[6,9,10] Normally, the arch of this blood vessel casts a rounded, ovoid or "almond" shaped density which rests in the angle between the trachea and right main bronchus. The shadow of the azygos vein becomes more prominent when in the course of cardiac decompensation a slight or moderate dilatation of this blood vessel takes place. Very pronounced enlargement of the azygos vein is, however, rarely encountered and seldom commented upon in the radiologic literature.

For this reason it was considered to be of interest to describe the following case in which acquired dilatation of the azygos vein reached such proportions that it simulated the presence of a mediastinal tumor. The description of this case may serve to briefly discuss some of the anatomic and roentgenologic features of the azygos vein under normal and pathologic conditions and emphasize its role as a collateral channel in superior and inferior vena cava obstruction.

REPORT OF A CASE

J.R.B., A-129116. This forty-nine year old white farmer was admitted to Grady Memorial Hospital in April of 1957 with symptoms and signs of congestive heart failure. The patient had been in good health until 1935 when there was sudden onset of right leg pain followed by rapid development of varicose veins and swelling of the legs as well as venous distention of the lower abdomen. Several years later he developed hypertension. Since 1951 he had been followed in the outpatient clinic with persistent albuminuria and elevated blood urea nitrogen. Retrograde and intravenous pyelograms in 1953 demonstrated a markedly hypoplastic nonfunctioning right kidney. It was thought that in 1935 he had a thrombosis of the right femoral vein with extension into the inferior vena cava and right renal vein with resulting atrophy of the right kidney and secondary hypertension. He was admitted to the hospital in 1955 and again in December of 1956 with mild congestive failure which responded promptly to treatment.

Physical examination at his last hospitalization showed normal blood pressure, evidence of cardiac decompensation and collateral venous circulation over the abdomen. The blood flow in the abdominal wall veins was craniad. No hemorrhoids were demonstrated.

Roentgenologic examination of the chest (Fig. 1*A*) soon after admission disclosed a round well circumscribed mass 3 cm. in diameter in the right superior mediastinum in the anatomic region of the azygos vein. This density had not been present on a previous chest roentgenogram five months before. Fluoroscopic examination during congestive failure did not show any intrinsic pulsations nor was there any change in its size with the Müller or Valsalva maneuvers. The roentgenogram in the supine position (Fig. 1*B*) did not modify the caliber of the described mass. Frontal and lateral laminagrams (Fig. 2, *A* and *B*) taken several days later again demonstrated this density which in the lateral view assumed a somewhat arch-like profile. The size of the density showed slight diminution. Repeat roentgenograms of the chest ten days after admission of the patient revealed a very pronounced decrease in the diameter of the observed density. In order to confirm the impression of marked dilatation of the azygos vein, intraosseous venography was performed. Contrast medium (15 cc. of 70 per cent urokon) was injected into the spinous process of the eleventh thoracic vertebra, leading to extensive filling of the vertebral venous plexus and opacification of the dilated azygos arch (Fig. 3). Subsequent upright roentgenograms (Fig. 4*A*) approximately four weeks after hospital admission failed to demonstrate the dilated

* From the Departments of Radiology, Emory University School of Medicine and Grady Memorial Hospital, Atlanta, Georgia.

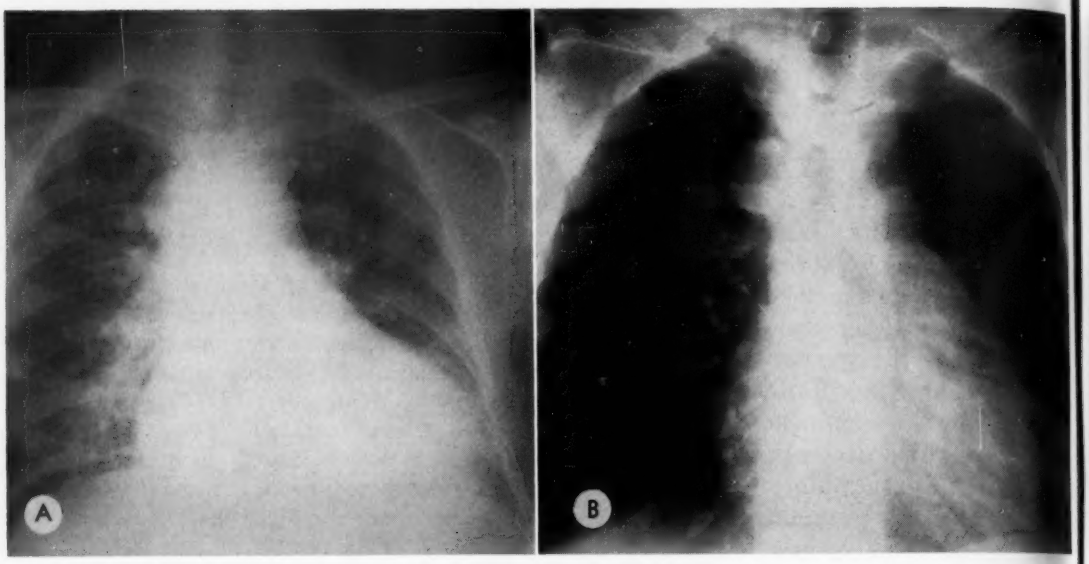

FIG. 1. (*A*) Upright roentgenogram of the chest at time of admission in severe congestive heart failure. Observe the well circumscribed rounded density in right superior mediastinum, cardiomegaly, and pulmonary congestion. (*B*) Supine roentgenogram of chest. The mass rests in the right tracheobronchial angle in the anatomic region of the azygos vein.

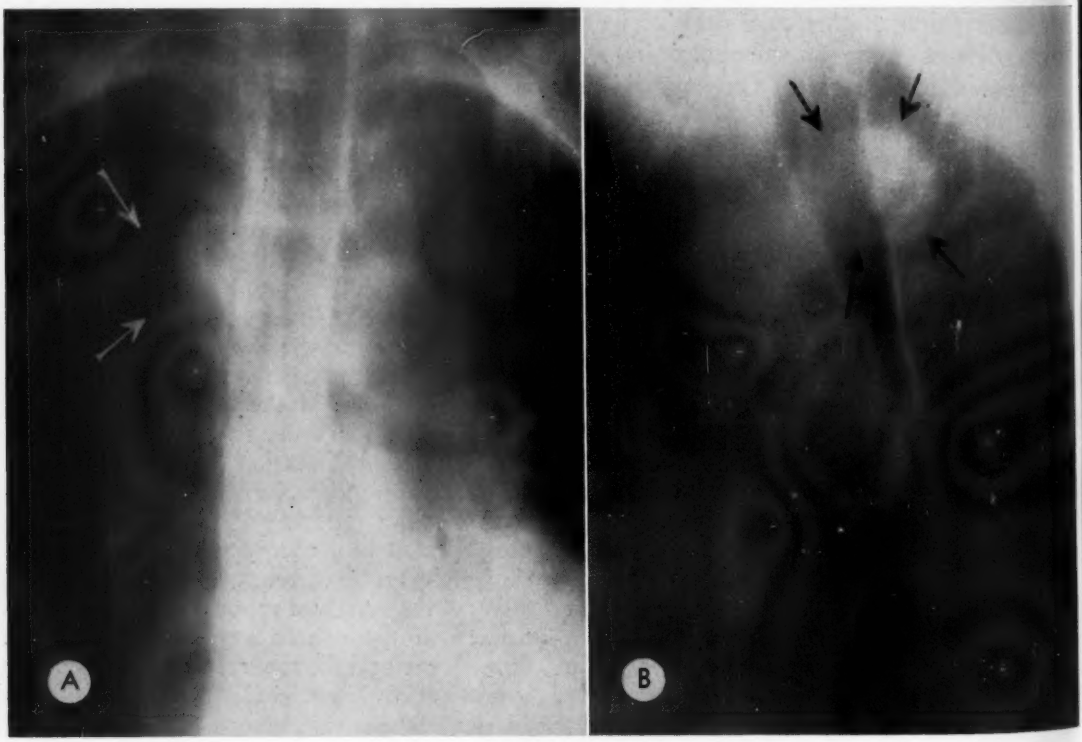

FIG. 2. Frontal (*A*) and right lateral (*B*) laminagrams of the chest reveal the arch-like profile of the mediastinal shadow.

Fig. 3. Intraosseous venogram. The contrast substance is well concentrated in the tracheobronchial angle mass, identifying it as the azygos vein (arrows).

azygos vein. However, in supine position the azygos vein was still clearly visible, measuring 16 mm. in diameter (Fig. 4*B*).

DISCUSSION

In a review of the literature Stauffer *et al.*[10] discuss the roentgenologic identification and catheterization of the normally situated arch of the azygos vein. Its appearance on frontal roentgenograms of the chest has been described as a "pumpkin seed" or "almond" shaped shadow adjacent to the right tracheobronchial angle. Fleischner and Udis[6] found that in normal individuals the width of the azygos vein shadow, including that of tracheal wall, rarely measured more than 5 mm.

The shadow of the azygos vein becomes more prominent during the Müller maneuver and diminishes in size during the Valsalva maneuver. In upright position the azygos vein tends to be partially collapsed whereas in roentgenograms in recumbent position it appears more fully

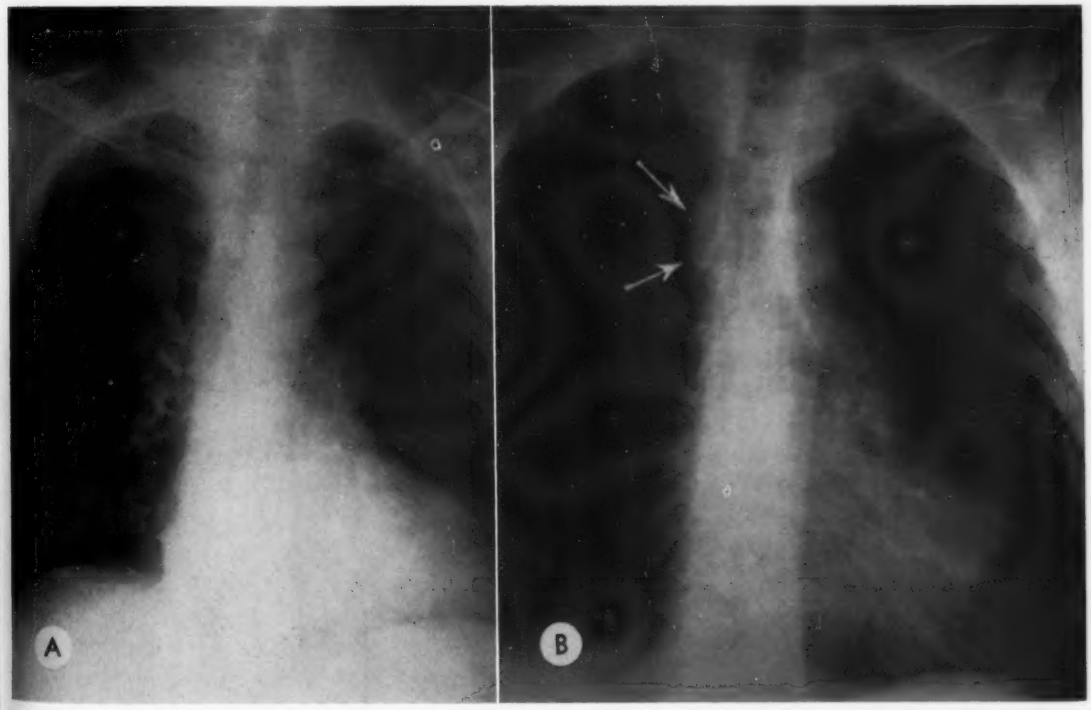

Fig. 4. (*A*) Upright roentgenogram of the chest approximately one month after initial roentgenogram. Note absence of mediastinal mass and decrease in heart size with clinical correction of cardiac decompensation. (*B*) Supine chest roentgenogram. The shadow of the azygos vein remains visible, but has decreased considerably in size (arrows).

distended. The cross section of the azygos vein is usually clearly visualized on frontal laminagrams of the right pulmonary hilus in supine position. These variations in the diameter of the azygos vein image, with change in position and intrathoracic pressure, may be helpful in distinguishing azygos vein dilatation from an enlarged mediastinal lymph node or tumor.

The most common cause of abnormal dilatation of the azygos vein is congestive heart failure. Fleischner and Udis,[6] studying the appearance of the azygos vein in patients with various cardiac disorders, noted that the width of the azygos vein was often increased in heart failure but rarely exceeded 13 mm.

In the presence of faulty development of the inferior vena cava, particularly of its suprarenal and hepatic segments, the azygos vein may represent a principal channel of venous return from the lower parts of the body to the heart. Malformations of this type have been described as aplasia or hypoplasia of the inferior vena cava with azygos drainage.[2,5] In the presence of this abnormality the dilated azygos arch is readily visible as a rounded density in the right tracheobronchial angle. Conceivably, cases of idiopathic dilatation of the azygos vein[9] may be related to such a congenital malformation.

In acquired obstruction of the superior or inferior vena cava or of the portal vein, the azygos system may afford one of the main routes by which venous circulation is maintained (Fig. 5). Under these circumstances the azygos system forms a collateral venous trunk parallel to the vertebral vein system as emphasized by Batson.[3] It is noteworthy that the azygos system and vertebral venous system have many anastomoses throughout the abdominal and thoracic region.

Stauffer and his associates[10] in their group of patients found the azygos vein largest in 2 individuals with occlusion of the inferior vena cava. It should be noted that in spite of the large shadow in the supine position there was absence of the azygos density in the conventional upright roentgenogram of the chest. Bronte-Stewart and Goetz[4] reported a case of Budd-Chiari's syndrome with unusual prominence of the right heart border which on venography was proved to be a large varicose azygos vein. Only rarely have cases been described in which dilatation of the azygos or hemiazygos vein assumed proportions so as to simulate the appearance of a mediastinal mass.[7,8,9]

Interest as to the size of the azygos vein shadow in patients with inferior vena cava ligation prompted us to study 6 patients in whom this operation had been performed one to ten years previously. The conventional upright chest roentgenogram appeared normal and there was likewise no significant increase in the size of the azygos shadow in the recumbent position or in the Müller maneuver. A barium swallow of these patients did not disclose any evidence of esophageal varices. The findings in these clinically normal patients would seem to indicate that although there may be increased blood flow in the azygos system after caval ligation, it could not be demonstrated by conventional roentgenologic procedures.

The case described in this paper is of

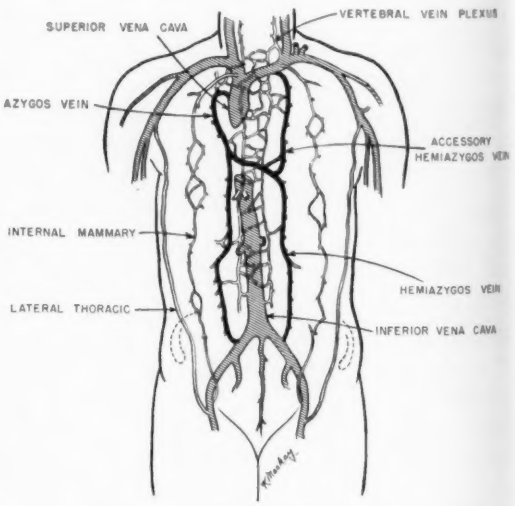

Fig. 5. Schematic diagram of venous circulation collateral to vena cava system. The azygos and hemiazygos systems are indicated in black.

particular interest because of the magnitude of azygos vein distention. Its cross sectional diameter measured slightly over 3 cm. in the initial upright chest roentgenogram whereas in congestive heart failure the azygos shadow rarely exceeds 13 mm. Several factors were apparently responsible for the aneurysmal size of the azygos vein. The rapid return of this blood vessel to normal size concomitant with the patient's clinical recovery justifies the hypothesis that cardiac decompensation was of primary importance. Occlusion of the inferior vena cava in this patient resulted in an increased blood flow through the azygos system. Conceivably, the thrombotic process involved other collateral venous channels normally responsible for venous return, imposing an additional workload on the azygos and hemiazygos system. Lastly, a variant of the many anomalous patterns of systemic venous return cannot be entirely dismissed.[1] One should also keep in mind that the location and length of the obstructed caval segment may, to some extent, determine the pattern and extent of the collateral venous circulation.

During the last two decades a relatively large number of vena cava ligations have been performed in patients with thrombophlebitis and phlebothrombosis, often in younger age groups. The development of heart disease with failure among these patients may result in a more frequent occurrence of the phenomenon described in this paper. When the radiologist encounters a mass in the anatomic region of the normally situated azygos vein, fluoroscopic examination and roentgenograms of the chest in upright, recumbent, and Trendelenburg positions as well as performance of the Valsalva and Müller maneuvers may give valuable clues as to its exact nature. It should be emphasized, however, that in our patient, while in severe congestive heart failure, no significant change could be demonstrated in the size of the azygos vein shadow in upright and recumbent roentgenograms. Conceivably, the azygos vein approached maximum distensibility in the upright position. Diminution of the azygos shadow as heart failure subsides may be of considerable diagnostic significance. Under these circumstances angiocardiography and intraosseous phlebography will be rarely necessary for a definite roentgenologic diagnosis.

SUMMARY

1. A case of thrombosis of the inferior vena cava is presented in which with development of cardiac decompensation the azygos vein dilated to such a degree as to simulate a mediastinal tumor.

2. The roentgenologic features of the azygos vein in normal and pathologic conditions are briefly discussed, and its role as a collateral channel in superior and inferior vena cava obstruction is emphasized.

3. A study of 6 patients in whom inferior vena cava ligation had been performed one to ten years previously revealed no visible increase in the size of the azygos shadow. It appears likely that cardiac decompensation was a major factor contributing to the huge dilatation of the azygos vein in the described case.

W. H. Shuford, M.D.
Emory University
69 Butler Street, S.E.
Atlanta 3, Georgia

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Vol. 80, No. 2

ABNORMAL MEDIASTINAL SHADOWS CAUSED BY THE TORTUOUS THORACIC AORTA*

By ITARU OHARA, M.D., *and* AIICHI TANNO, M.D.
SENDAI, JAPAN

AT PRESENT, mediastinal tumors can be operated upon with low mortality and morbidity. As these tumors show diversified shadows on the posteroanterior chest roentgenogram, careful examination is required before surgical removal is attempted. Hitherto, the most common cause of mediastinal enlargement by large vessels was thought to be due to aneurysm of the aorta or aortitis.[1] Recently, two other causes have been added. One is coarctation[3] and the other is the so-called pseudocoarctation or kinking of the aorta.[2] The latter appears as a rounded bulging from the mediastinum into the left lung field, separate from the aortic knob. Hypertension in the upper extremities or notched ribs are not present in these cases. Usually, pseudocoarctation can be differentiated from other mediastinal masses by an oblique or lateral roentgenogram, but occasionaly angiocardiography is necessary for its confirmation.[4,9]

Not uncommonly, tortuosity and elongation of the thoracic aorta are encountered in persons of advanced age having arteriosclerosis or hypertension.[7] These can be diagnosed without difficulty. We have, however, observed on the conventional chest roentgenograms unusual shadows of this vessel simulating a mediastinal tumor in 3 cases. These cases are reported in order to call attention to the roentgen appearance in such instances.

REPORT OF CASES

CASE I. T.T., a sixty-seven year old male, was admitted to the University Hospital with left abdominal pain and dysphagia. The patient stated that he had had a chronic cough since a diagnosis of pneumonia was made a year previously and had noticed blood in his sputum recently. He also complained of a distressing dull but occasionally piercing neuralgic pain in the left flank of five months' duration and difficulty in swallowing for a period of twenty days. Physical examination revealed an undernourished man with anemia. Both radial arteries were sclerotic, the pulse rate was 80 in sinus rhythm and the blood pressure 110/60. The serum Wassermann reaction was negative. Heart sounds were clear.

The posteroanterior roentgenogram of the chest revealed a bilateral rounded homogeneous enlargement of the mediastinal shadow (Fig. 1). The left anterior oblique roentgenogram showed no mediastinal tumor but a dilated and posteriorly displaced descending thoracic aorta, of which the lower one-third was found merging into the cardiac shadow. The general outline of the thoracic aorta was demonstrated by posteroanterior laminagrams taken at 9, 11, 13 (Fig. 2) and 15 cm. from the back. The right mediastinal shadow was caused by the ascending aorta, while the left mediastinal shadow was due to the laterally displaced descending aorta. The lower part of the descending aorta was seen traversing over the ninth thoracic vertebral body, constituting part of the shadow of the right cardiac border. Later, this finding was confirmed by serial angiocardiography.

CASE II. H.T., a sixty-two year old male, came to the University Hospital with a chief complaint of dysphagia. At the age of forty-nine, he suffered an attack of left hemiplegia. Three months prior to admission, he had noticed difficulty in swallowing solid food and was only able to take fluids. Recently, he began to expectorate bloody sputum. Physical examination revealed an undernourished man in anxiety. Pulse rate was 68 in sinus rhythm and the blood pressure 146/102. The serum Wassermann reaction was not known. A slight degree of left hemiplegia was found. The heart was enlarged to the left but no abnormal cardiac murmur was detected. The electrocardiogram

* From the Surgical Clinic of the Tohoku University, Faculty of Medicine, Sendai, Japan (Director: Prof. S. Katsura). Part of the paper was read before the Fifth Meeting of the Japanese Circulation Society, the Tohoku Hokkaido Chapter, Sendai, February 8, 1958.

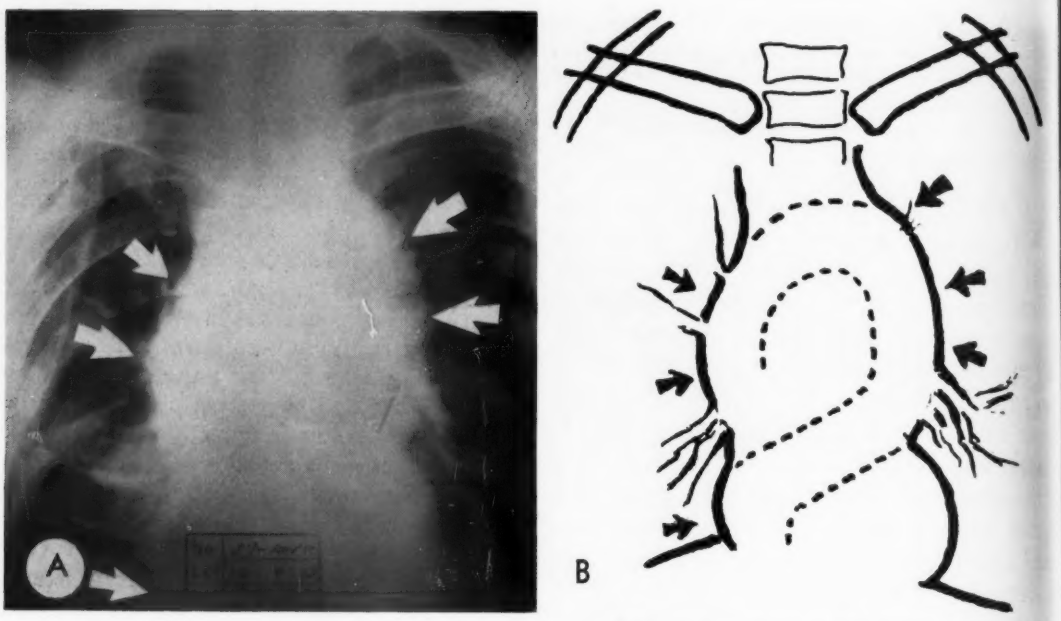

FIG. 1. Case I. (*A*) Posteroanterior roentgenogram shows bilateral enlargement of the mediastinum (white arrows). (*B*) Diagram shows dilatation and tortuosity of the ascending and descending thoracic aorta (dotted lines) causing the enlargement.

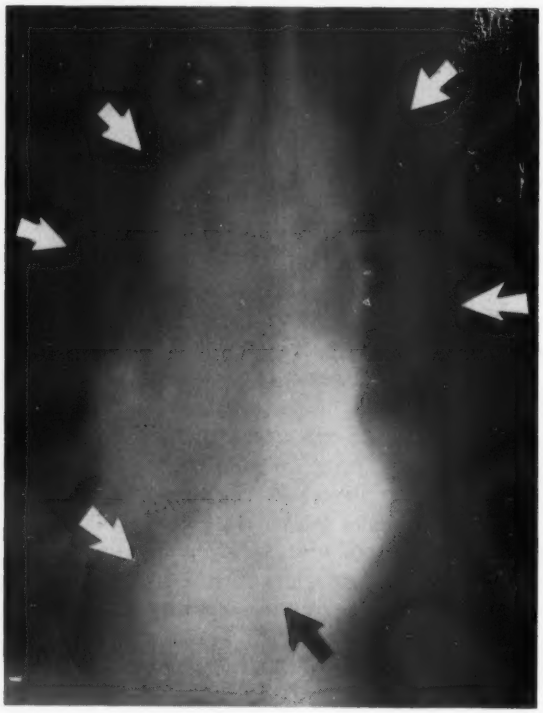

FIG. 2. Case I. Laminagram at 13 cm. outlines the course of the descending thoracic aorta (white and black arrows).

showed a reversed T in lead I and a depression of the S-T segment in other limb leads, suggesting myocardial damage.

The posteroanterior roentgenogram revealed a homogeneous mass with a rounded contour in the right mediastinum (Fig. 3). A chest roentgenogram exposed at a higher voltage showed that the descending thoracic aorta turned to the right below the aortic knob (Fig. 4). One-third of its course was in the right chest instead of along the spinal column. The right border of the heart was within the vascular shadow. The right anterior oblique roentgenogram showed a clearly visible shadow over the vertebral column (Fig. 5). Obviously, the mediastinal mass was due to the descending thoracic aorta.

CASE III. N.Y., a sixty-seven year old male, entered the University Hospital with dysphagia and loss of weight as the chief complaints. Three months previously, he had noticed difficulty in swallowing. He was transferred from the Medical Clinic to the Surgical Service for operation of a carcinoma of the lower esophagus. Physical examination revealed a slightly undernourished man of average height. The

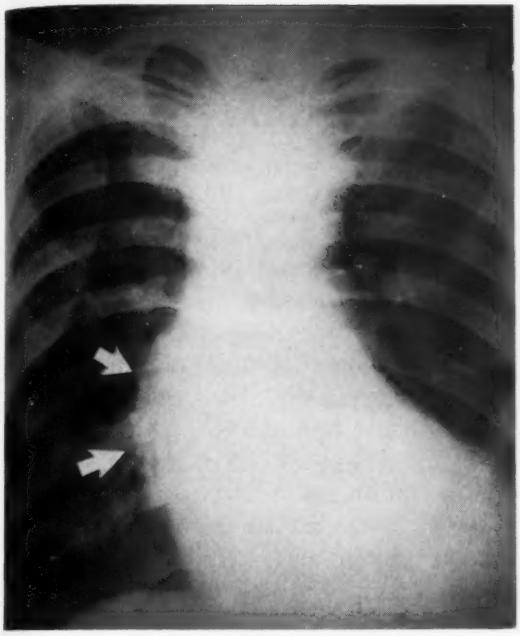

Fig. 3. Case 11. Chest roentgenogram shows mediastinal enlargement to the right (white arrows).

pulse rate was 58 in sinus arrhythmia. The electrocardiogram showed atrial fibrillation and flattening of the T wave in all leads, suggesting myocardial damage. The blood pressure was 115/80. No abnormality of the chest cage or superficial venous dilatation was observed. The Wassermann reaction was negative. No abnormal heart sounds were audible.

A conventional chest roentgenogram in the posteroanterior view showed enlargement of the mediastinal shadow in both directions and a mass below the right clavicle (Fig. 6). The left anterior oblique roentgenogram demonstrated dilatation of the ascending and upper third of the descending aorta, while the lower half was seen disappearing behind the cardiac silhouette. A posteroanterior roentgenogram taken at a higher voltage clearly demonstrated that the right mediastinal shadow was caused by the ascending aorta and the left mediastinal shadow by the descending aorta (Fig. 7). Part of the descending aorta which crossed the spinal column mimicked the outline of the right heart. The esophagus was found coursing along the

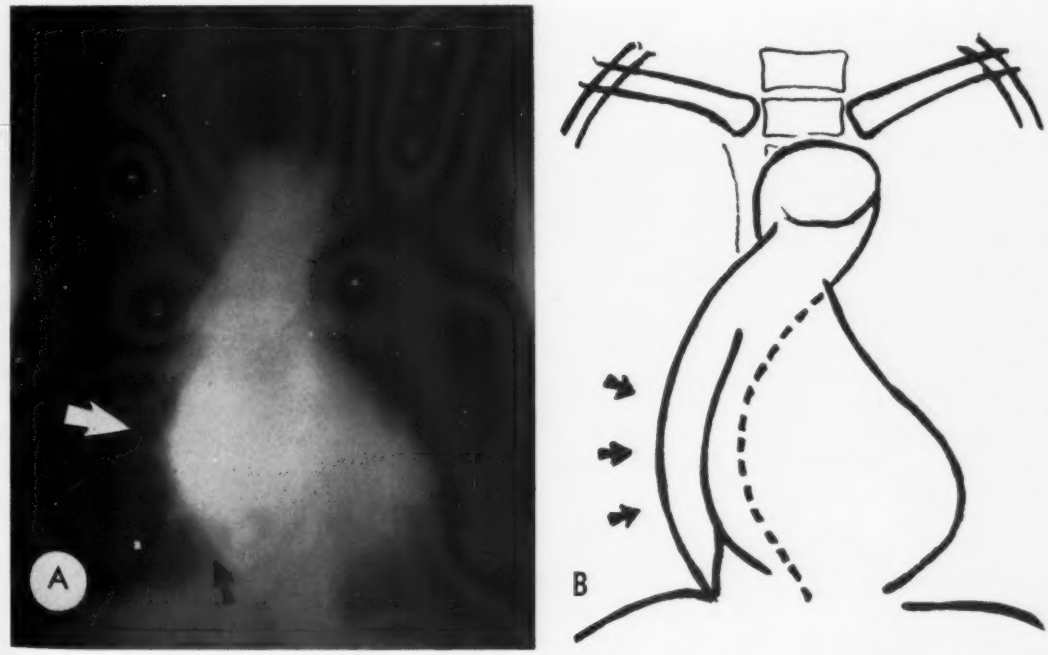

Fig. 4. Case 11. (*A*) Chest roentgenogram made with a higher voltage exposure. Posteroanterior view. The descending thoracic aorta crosses over the dorsal vertebrae (white arrow) and superimposes on the right border of the heart (black arrow). (*B*) The descending thoracic aorta (black arrow) takes a course to the right (dotted lines and black arrows).

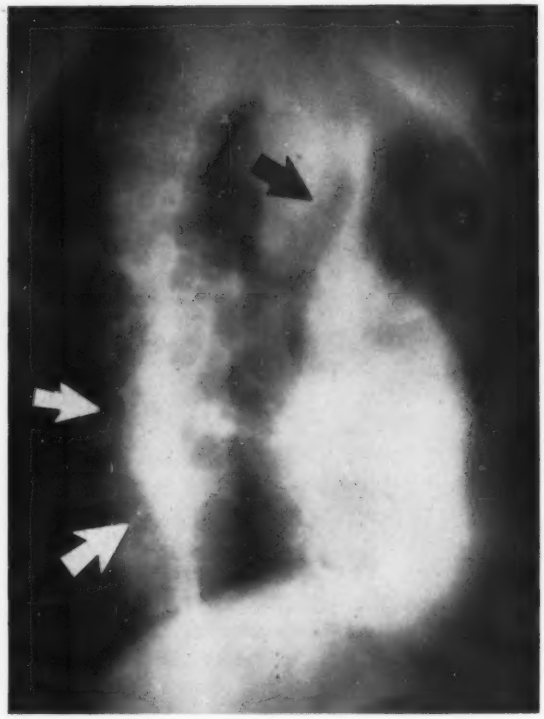

Fig. 5. Case II. In the right anterior oblique view, the trachea is displaced anteriorly (black arrow). The descending thoracic aorta crosses over the spine (white arrows) and the right border of the vessel is in the right chest.

descending aorta, first right to the spine, then left to the spine at the esophageal hiatus. The unusual course of the aorta was confirmed by palpation during the surgical intervention for carcinoma of the lower esophagus.

DISCUSSION

Tortuosity and elongation of the abdominal aorta are demonstrated by abdominal aortography and usually constitute no diagnostic problems. However, if these anomalies are present in the thoracic aorta, an abnormal shadow on routine chest roentgenograms may be observed, which should be differentiated from the symptomless mediastinal tumors.[5] A tortuous aorta has a round contour like that of lymphoma (Hodgkin's type), cysts, various benign and malignant tumors, or aneurysms.[3,10] Traction of the tortuous aorta at the ligamentum arteriosum is known to produce a tumor-like appearance.[12] Roentgenograms in various views together with a barium study of the digestive tract and bronchography are required to establish a differential diagnosis. In our first case, an overpenetrated chest

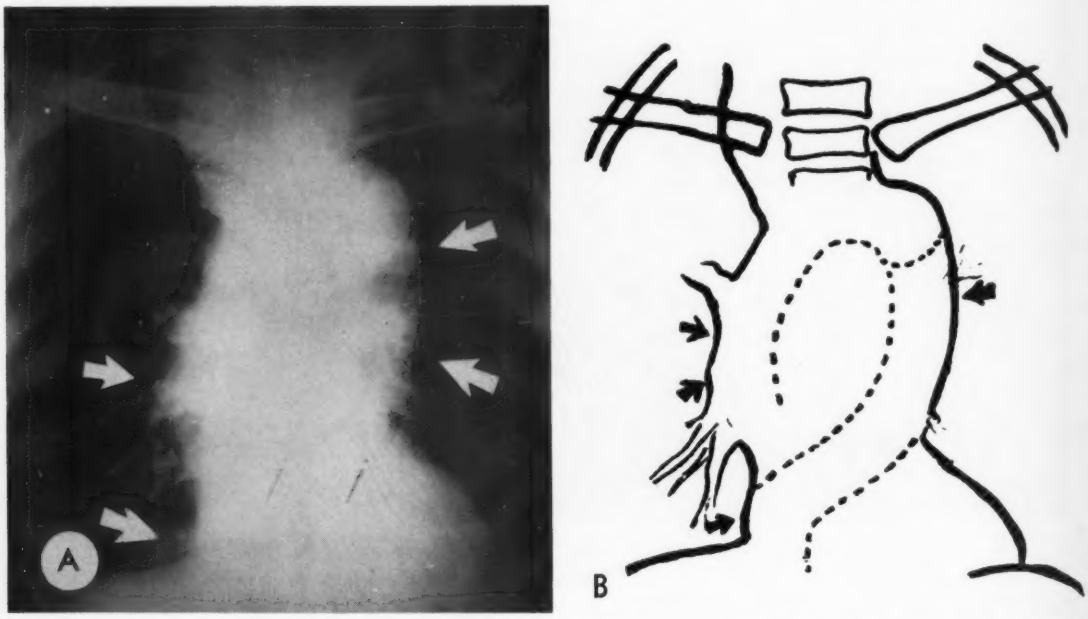

Fig. 6. Case III. (*A*) Posteroanterior roentgenogram shows enlargement of the mediastinal shadow in both directions (white arrows). The right lower border of the descending aorta disappears behind the heart shadow (right lower arrow). (*B*) The course of the aorta is shown by arrows and dotted lines.

roentgenogram, together with some other diagnostic tests, was helpful in excluding mediastinal tumor. Laminagraphy was found to be a simple and useful technique in visualizing the course of the aorta and is recommended as a diagnostic aid. A review of the literature reveals that in several cases angiocardiography has been employed to arrive at the correct diagnosis.[4,11] Undoubtedly, this is the best procedure for obtaining conclusive evidence.

In rare instances, the descending aorta is said to cross over the vertebral column into the right chest.[7] Roesler and White[6] described such a case in which the vessel strikingly projected into the right lung field and made a differential diagnosis from aneurysm of the ascending aorta very difficult. Case II reported above is less advanced, but does have similar characteristics. As already suggested by Roesler and White,[6] an overpenetrated roentgenogram helps in visualizing clearly the contour of the aorta and eliminating other confusing shadows. This was demonstrated in our second and third case. Unusual mediastinal shadows are said to occur at two points of the fixation of the elongated aorta *i.e.*, heart and diaphragm. Vessel anomalies, such as right-sided aorta should also be seriously considered.[8]

FIG. 7. Posteroanterior chest roentgenogram made with a higher voltage exposure. The original roentgenogram faintly delineated the ascending aorta where the right upper arrow points. The trachea is slightly displaced to the right (black arrow). The descending thoracic aorta crosses over the spinal column into the right chest (white arrow).

SUMMARY

Three cases of dilatation and elongation of the thoracic aorta causing rounded mediastinal shadows are reported. All patients were over sixty years of age and had arteriosclerosis although the systolic blood pressure was below 150 mm. of mercury. Two cases showed negative Wassermann reactions and in the third it was not determined. The diagnosis of mediastinal tumor was seriously considered in all three cases. In each instance, the conventional roentgenographic procedures, which seemed indicated, were carried out. Of these, posteroanterior roentgenography with higher voltages and laminagraphy were most helpful in establishing that the aorta was the cause of the abnormal shadows. In all cases, the descending aorta was found crossing over the vertebral column to the right side. In two cases, the descending aorta formed part of the right border of the heart.

Due attention should be paid to blood vessel abnormality when interpreting mediastinal masses on conventional roentgenograms.

I. Ohara, M.D.
Tohoku University Hospital
Sendai, Japan

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ROENTGEN DIAGNOSIS OF ACUTE AND CHRONIC TRAUMATIC ANEURYSM OF THE THORACIC AORTA*

By ISRAEL STEINBERG, M.D., *and* JOHN A. EVANS, M.D.
NEW YORK, NEW YORK

ANEURYSMS of the thoracic aorta due to blunt trauma occur more frequently than suspected. This is because increasing speed of the present vehicular era has made accidents commonplace. Formerly, the diagnosis of aortic rupture during life was rarely made; multiple fractures and internal injuries associated with profound shock overshadowed the thoracic injuries and the chest was overlooked. Hollingsworth *et al.*,[8] Wyman,[21] and Goyette *et al.*[5] were among the first to emphasize the importance of routine serial roentgenograms made at the bedside after accidents for diagnosis of mediastinal hematoma and thoracic aortic aneurysm. Subsequently, the possibility of surgical intervention in aortic rupture as a life-saving measure was stressed[9,18] and the importance of the chest roentgenogram after injury even in the absence of visible thoracic injury became accepted generally.

Rupture of the thoracic aorta due to blunt trauma occurs most frequently at the top of the aorta (Fig. 1).[11] This is explained by the physical forces which are concerned with deceleration acting with undue stress on the aorta, producing a tear just beyond the point of fixation by the brachiocephalic arteries and the insertion of the ligamentum arteriosum. Strassmann,[20] in a classic report of 72 cases of aortic rupture among 7,000 autopsies from the files of the Chief Medical Examiner's office of the borough of Manhattan of New York City during the years 1936 to 1942, found that the majority of the cases occurred at this site; 20 per cent occurred at the ascending aorta just above the cusps; and 20 per cent in the descending aorta from the isthmus to the diaphragm. In all but 8 subjects, the aortic rupture was through-and-through and involved all the layers of the aorta. In the 8 cases, rupture was incomplete; the adventitia being preserved and dissected from the medial layer by thrombus. When all the layers of the thoracic aorta are torn, fatal hemorrhage usually results. In a few, especially when the adventitia is spared, there may be organization of the thrombus with a connective tissue wall and formation of a false aneurysm with recovery. In the chronic stage, deposition of calcium in the aneurysm very often takes place.

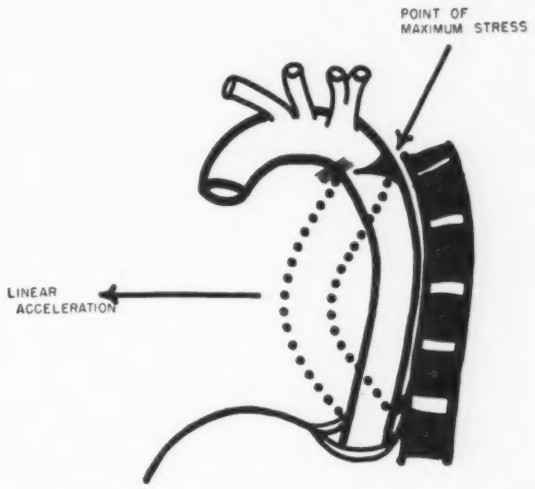

FIG. 1. Diagrammatic representation of the mechanism of laceration of the healthy aorta by indirect violence. (Reproduced with permission of Dr. W. G. Rice[11] and *The Journal of the American Medical Association.*)

Roentgenographically, the course of events following acute aortic injury is illustrated in Figure 2. At first there is widening of the mediastinum with obscuration of the arch, often with associated

* From the Department of Radiology, The New York Hospital-Cornell Medical Center.
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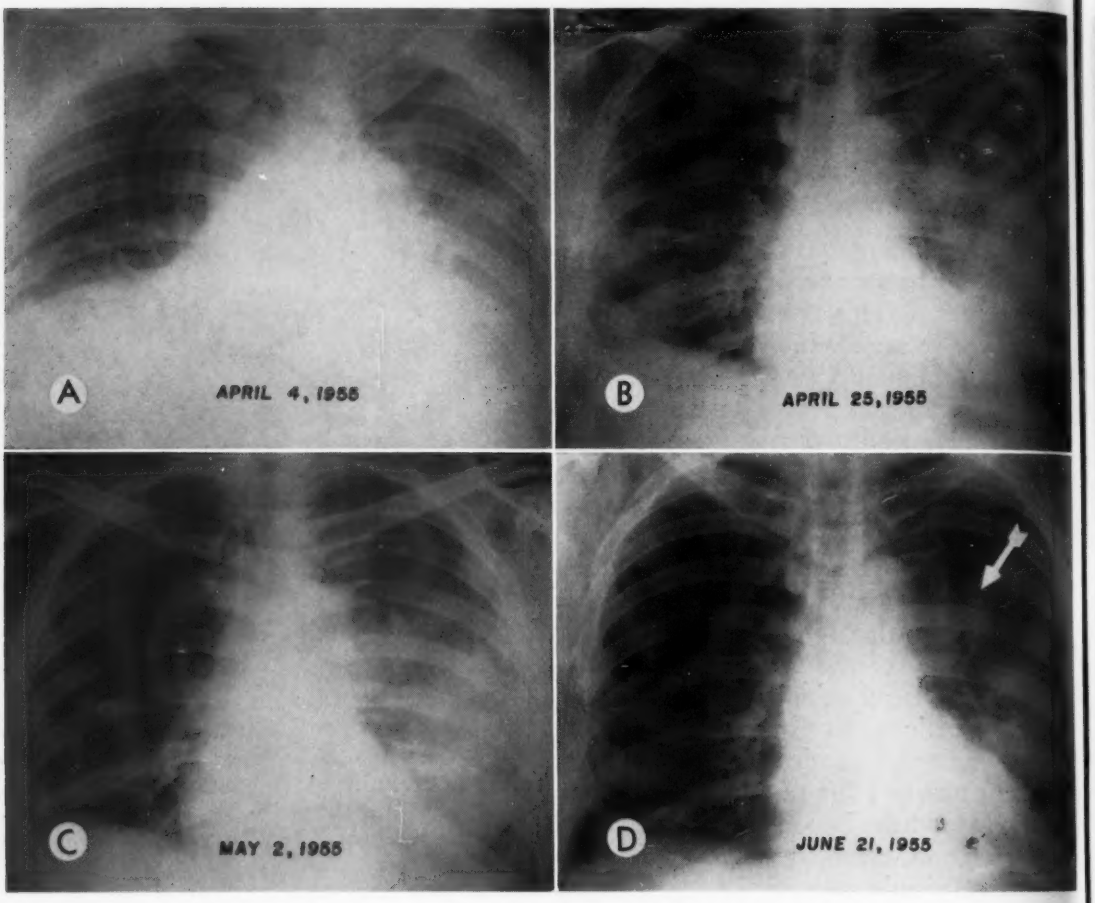

FIG. 2. Roentgenograms of a forty-five year old man who had a steering wheel injury in an automobile collision. (*A*) Frontal bedside roentgenogram shows widening of the supracardiac area and left pleural effusion. (*B*) Three weeks later, there is a vague paraaortic knob density with haziness in the lower half of the left lung field. The right lung shows an effusion in the costophrenic angle and thickening of the interlobar pleura. (*C*) A month after the accident, rounding of the mass adjacent to the aortic knob is becoming evident. (*D*) Eleven weeks after injury, there is subsidence of the right lung densities. The masses adjacent to the aortic knob (arrow) and at the left lower lung field, although smaller, are more rounded and distinct.

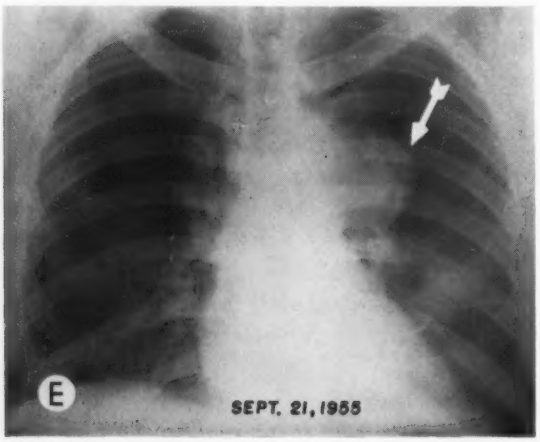

FIG. 2. (*E*) Six months after trauma, the mass adjacent to the aortic knob (arrow) has become well demarcated.

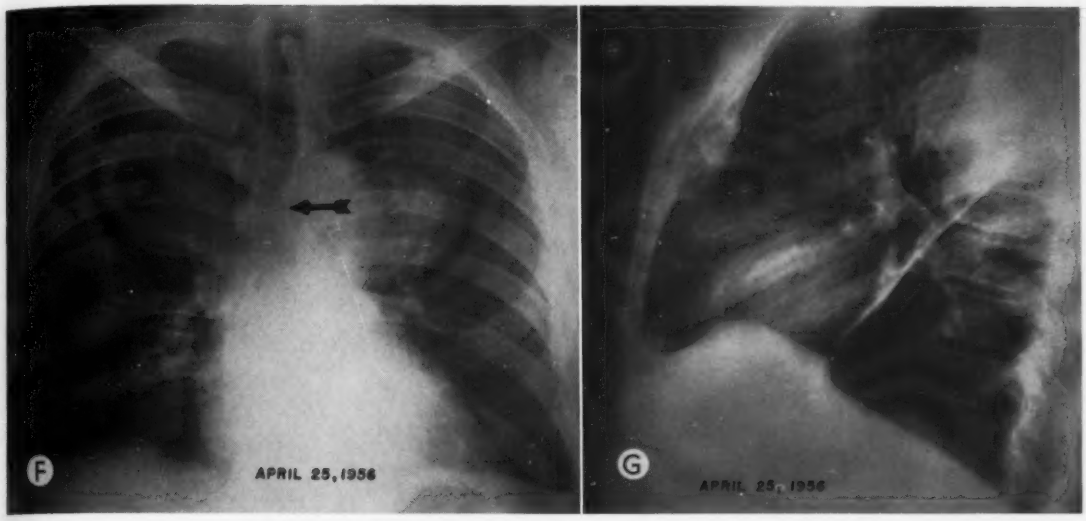

Fig. 2. (*F*) One year after the accident, the aneurysm is dense, large and deviates the esophagus (arrow). (*G*) Lateral view shows the fractured sternum. The aneurysm at the top of the descending aorta is dense and there is thickening of the interlobar pleura.

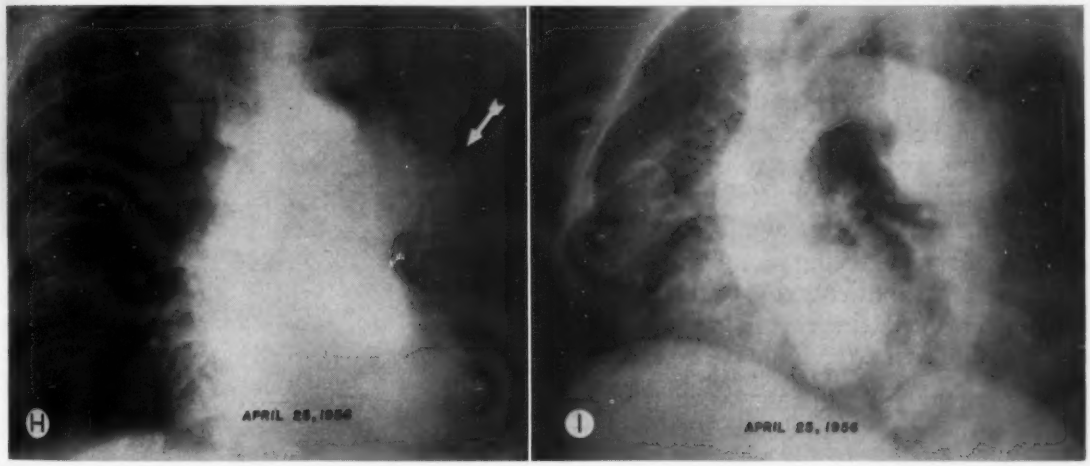

Fig. 2. (*H*) Frontal angiocardiogram reveals the saccular aneurysm lined by a thick wall of thrombus (arrow). (*I*) Left anterior oblique angiocardiogram shows the sacciform aneurysm at the top of the descending aorta arising beyond the left subclavian artery. (*H* and *I* reproduced with permission of *The New England Journal of Medicine*.[17])

pleural hemorrhage which produces opaqueness of the left lung (Fig. 2*A*). Subsequently, the mediastinal hematoma may subside but the left hemothorax persists (Fig. 2*B*). Within two to four weeks, a rounded rather vague bulge in the region of the aortic knob may become evident (Fig. 2*C*). Later, usually within two months, an aortic mass becomes more evident while the pleural hematoma subsides (Fig. 2*D*). Still later, after a few months, the outline of an aortic aneurysm becomes demarcated and more distinct (Fig. 2*E*). A year later (Fig. 2, *F* and *G*), the aneurysm appears large, dense and rounded. Very often the aneurysm may be nonpulsatile due to the surrounding thrombus (Fig. 2*H*). Calcium, sometimes in layers or diffusely deposited, is common in older aneurysms. The location of the aneurysm, at the top of

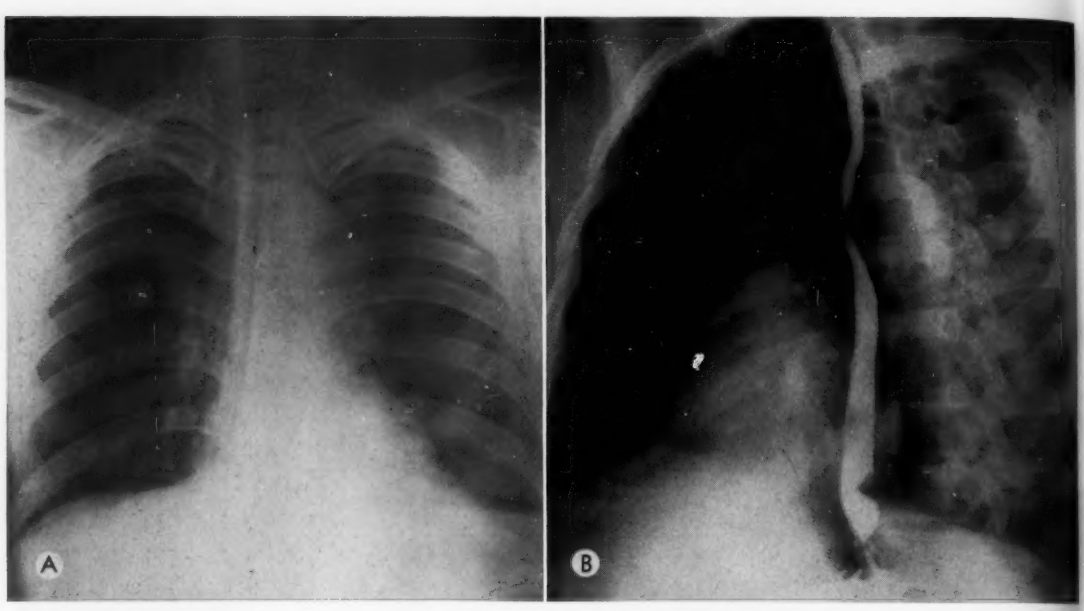

FIG. 3. Roentgenograms of a twenty-nine year old sailor who was injured in a jeep accident. (*A*) Frontal routine roentgenogram made five years after injury shows an aortic aneurysm adjacent to the aortic knob. There is obliteration of the left costophrenic sulcus. (*B*) Lateral esophagram reveals the dense aneurysm at the top of the descending aorta deviating the esophagus.

the descending aorta and the relation to the left subclavian artery are well illustrated by angiocardiography in the left anterior oblique position (Fig. 2*I*).

Chronic traumatic aneurysms of the thoracic aorta are being found more often in asymptomatic patients due to the increased popularity of routine roentgenographic surveys of the chest.[5,7,8] Four such cases have been recently encountered.[17] The first patient (Fig. 3, *A* and *B*) had been in an automobile accident five years earlier, and showed a mediastinal mass in the region of the aortic knob. The mass was pulsatile and deviated the esophagus (Fig. 3*B*). Angiocardiography confirmed the aneurysmal nature of the lesion. Resection of the aneurysm with the use of a heterologous shunt with hypothermia and replacement by a homograft was unsuccessful. The patient died of pulmonary edema twenty-four hours postoperatively.

Another patient (Fig. 4) was found to have a left mediastinal mass on a routine roentgenogram taken on admission to college and had a history of having been in an automobile accident at the age of four years. The mass was dense, rounded and pulsatile. A fine rim of calcium lined its posterior border (Fig. 4, *A* and *B*). Angiocardiography established the presence of a huge sacciform aneurysm which began just beyond the origin of the left subclavian artery (Fig. 4*C*).

The third patient was found after routine fluororoentgenography done during a cancer detection examination. Slight widening of the right superior mediastinum by a mass containing fine linear calcifications was recognized (Fig. 5*A*). As a result, detailed roentgen studies were made and disclosed displacement of the esophagus anteriorly and to the right by a rounded pulsatile mass (Fig. 5, *B*, *C* and *D*). The linear calcifications were especially well demonstrated in the oblique studies (Fig. 5, *C* and *D*). Angiocardiography (Fig. 5, *E–G*) showed a sacciform aneurysm at the top of the descending aorta, the favorite site of traumatic aortic aneurysms. Direct questioning then elicited the history of a serious automobile accident involving trauma to the chest twenty-one years earlier; thereby, establishing the etiology of the aneurysm.

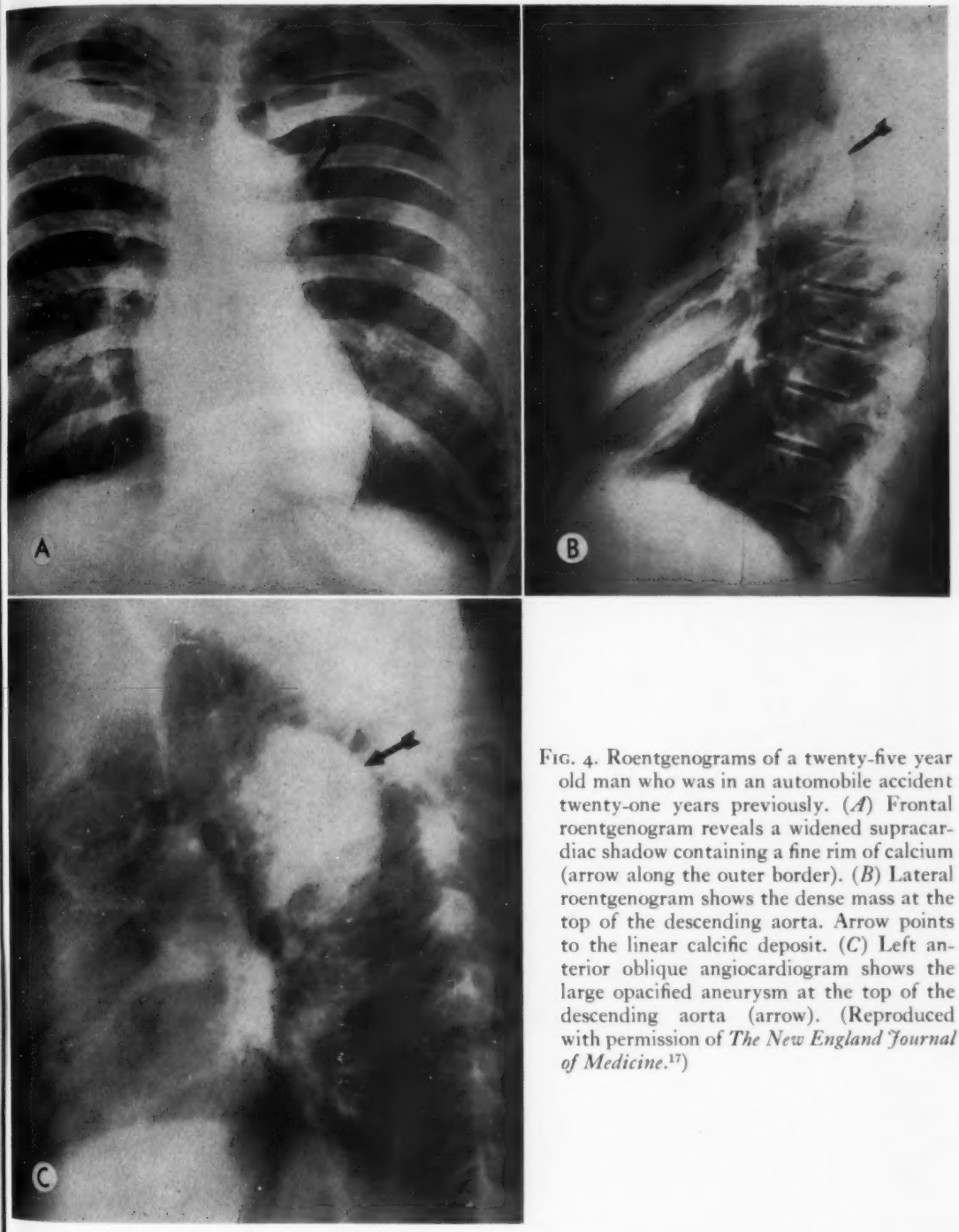

FIG. 4. Roentgenograms of a twenty-five year old man who was in an automobile accident twenty-one years previously. (*A*) Frontal roentgenogram reveals a widened supracardiac shadow containing a fine rim of calcium (arrow along the outer border). (*B*) Lateral roentgenogram shows the dense mass at the top of the descending aorta. Arrow points to the linear calcific deposit. (*C*) Left anterior oblique angiocardiogram shows the large opacified aneurysm at the top of the descending aorta (arrow). (Reproduced with permission of *The New England Journal of Medicine*.[17])

Syphilis was excluded by negative serologic studies.

The last patient (Fig. 6) during a routine roentgenoscopic examination of the chest was found to have a calcified mass in the region of the aortic knob. The patient refused hospitalization because she was asymptomatic. However, two years later, she developed vague lower back pains and was prevailed upon to enter the hospital.

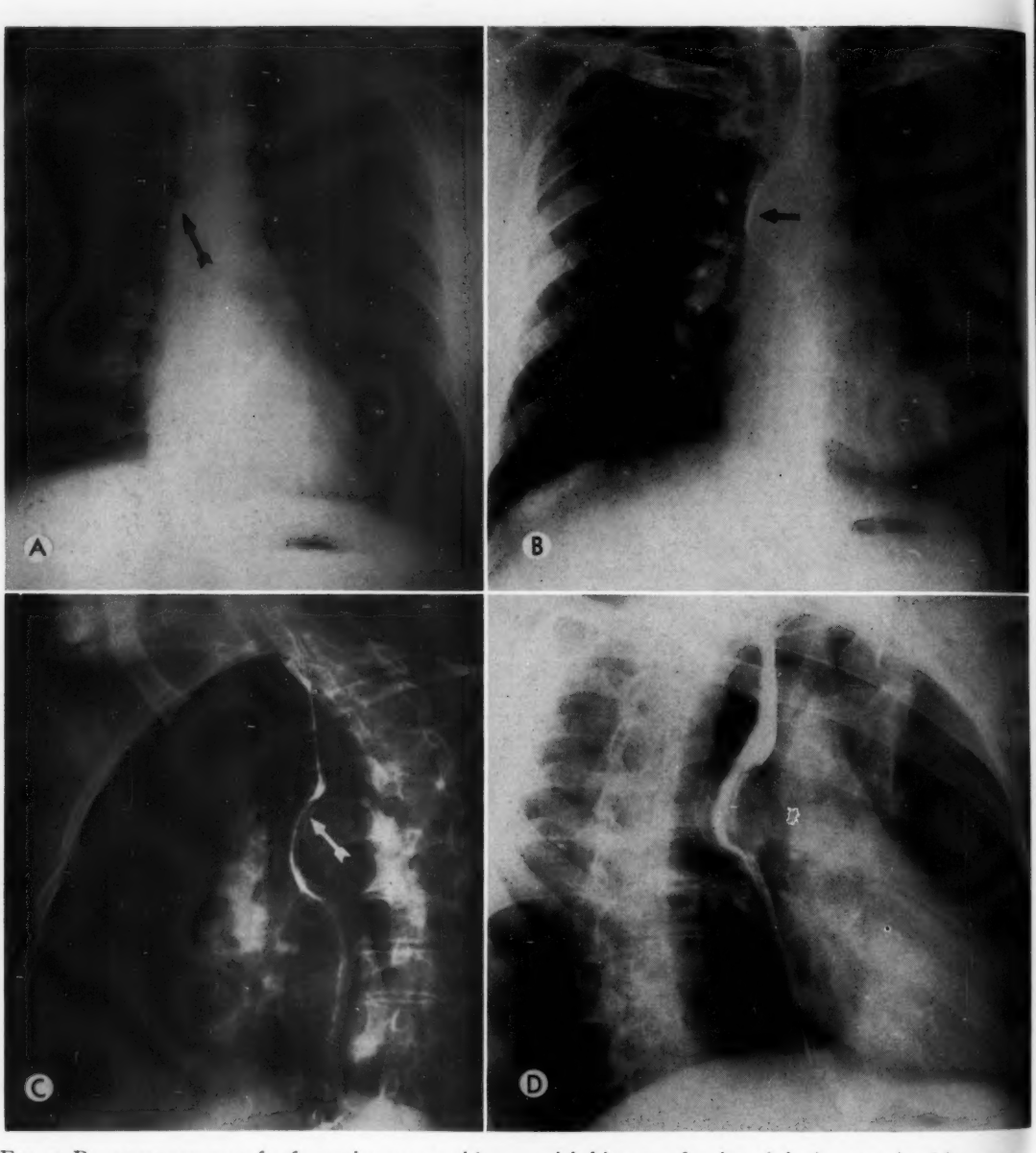

FIG. 5. Roentgenograms of a forty-three year old man with history of serious injuries sustained in an automobile accident twenty-one years earlier. (*A*) Frontal roentgenogram, made after a routine 70 mm. photoroentgenographic study, shows calcium (arrow) along the right superior mediastinal margin. (*B*) Frontal esophagram shows deviation of the esophagus by the mass. Arrow points to the rim of calcium in the mass. (*C*) Left oblique esophagram also reveals the calcium containing mass (arrow) deviating the esophagus. (*D*) Right oblique esophagram shows relations of the mass.

The chest roentgenograms (Fig. 6, *A* and *B*) disclosed a huge calcified nonpulsatile mass adjacent to the aortic knob producing forward displacement of the esophagus (Fig. 6*B*). Serologic tests for syphilis were negative. Angiocardiography (Fig. 6*C*) readily established the diagnosis of aortic aneurysm. Again, because of the characteristic location of the aneurysm at the top of the descending aorta, direct questioning uncovered the history of an automobile accident twenty-seven years earlier.

DISCUSSION

Aneurysms of the thoracic aorta following blunt trauma may be divided into the

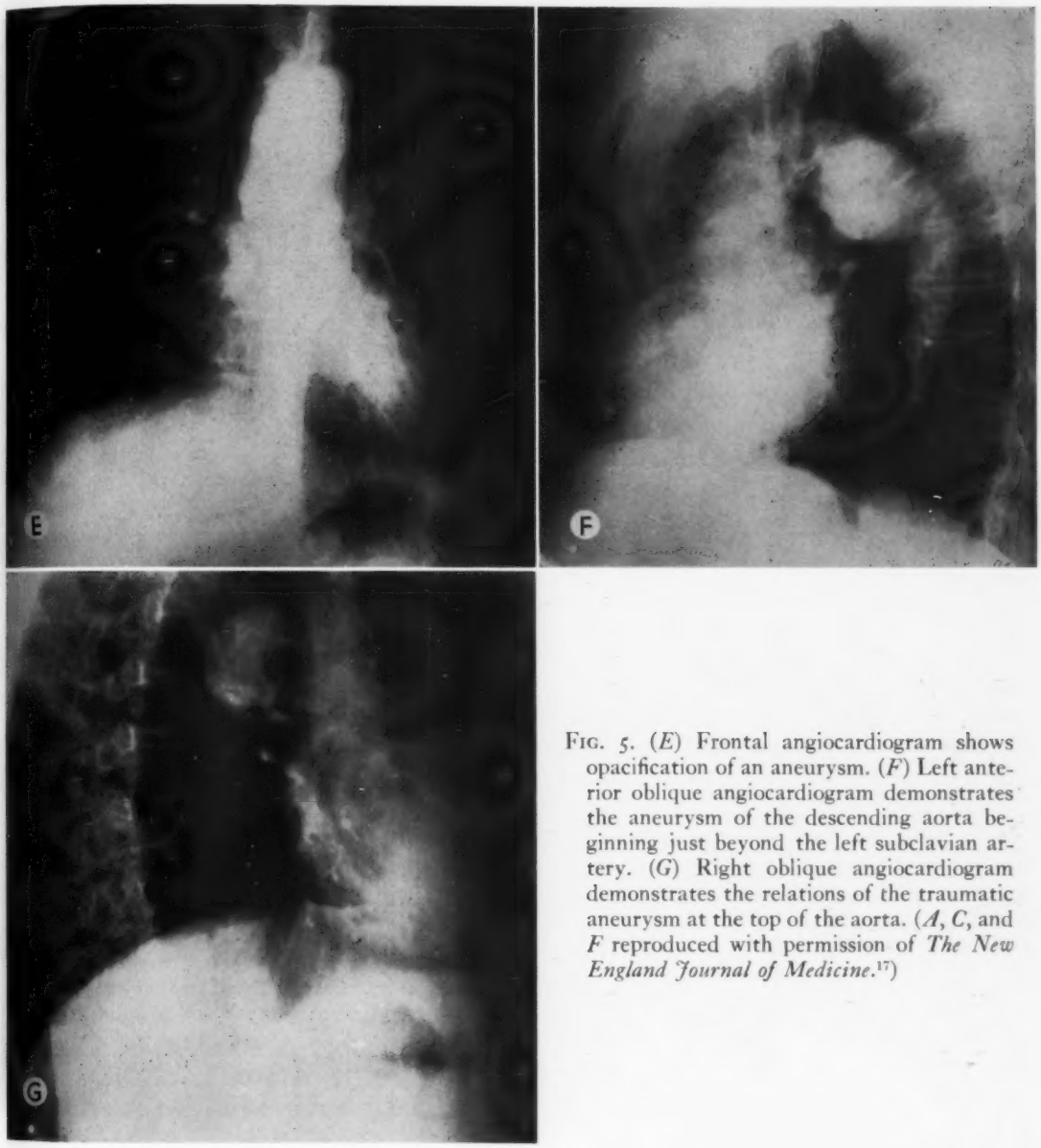

FIG. 5. (*E*) Frontal angiocardiogram shows opacification of an aneurysm. (*F*) Left anterior oblique angiocardiogram demonstrates the aneurysm of the descending aorta beginning just beyond the left subclavian artery. (*G*) Right oblique angiocardiogram demonstrates the relations of the traumatic aneurysm at the top of the aorta. (*A*, *C*, and *F* reproduced with permission of *The New England Journal of Medicine*.[17])

acute and chronic types. In the *acute* stage, unconsciousness, shock, multiple fractures and internal injuries may divert attention away from the thorax and result in overlooking aortic rupture. Recently several authors have stressed the importance of routine serial chest roentgenography following injuries. This has resulted in the diagnosis of aortic rupture during life and provided an understanding of the course of events in the patients that survive. At first, there is widening of the mediastinum and obscuration of the arch of the aorta, with fluid formation in the left chest due to mediastinal hematoma and hemothorax. Later, in a few days to three or four weeks, a vague bulge appears adjacent to and indistinguishable from the aortic knob; the aneurysm then becomes localized and distinct. Such was the course of events in a patient who was observed from the onset of injury (Fig. 2, *A–E*).

In 4 patients recently studied[17] and in many instances reported in the literature,[5,7]

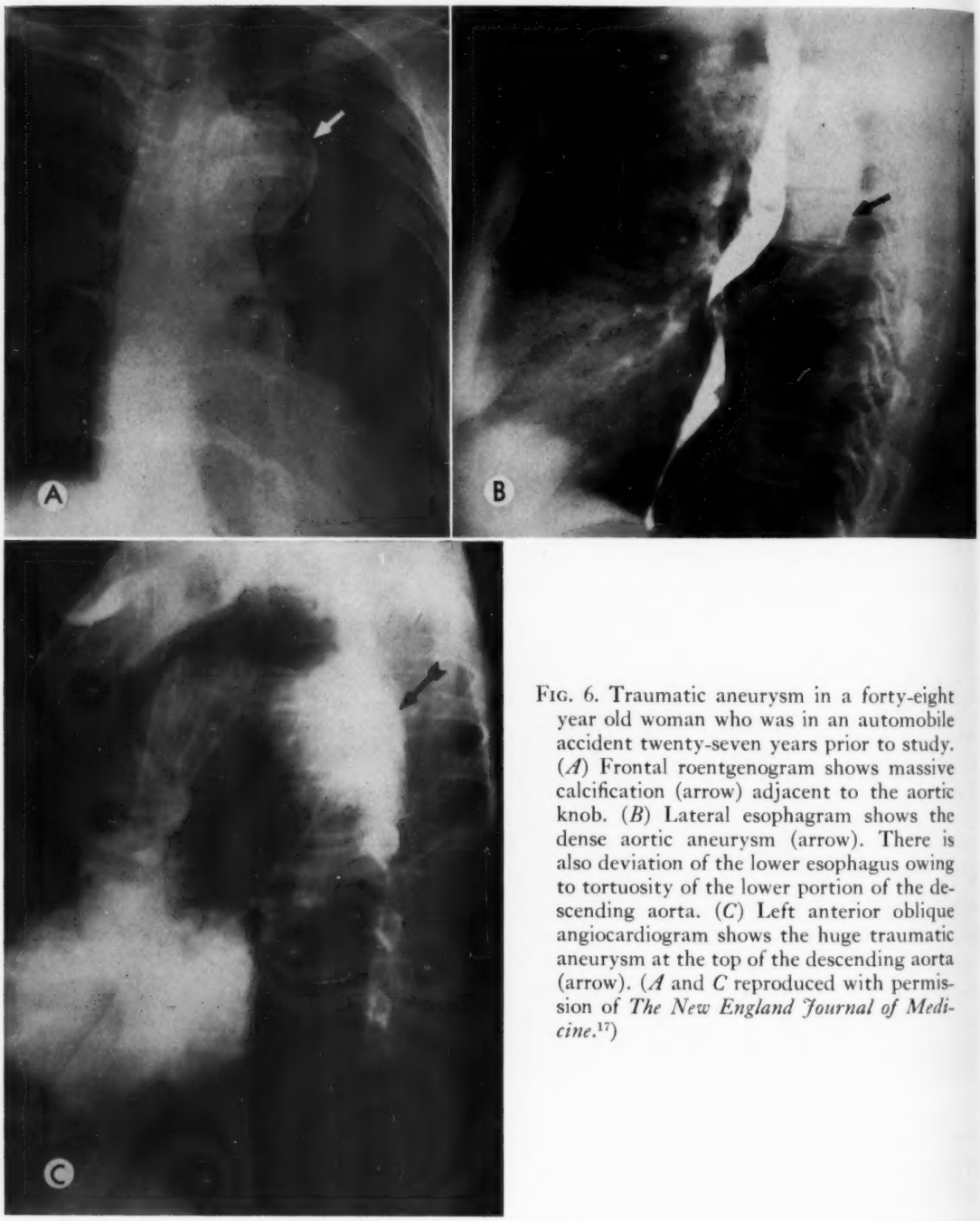

FIG. 6. Traumatic aneurysm in a forty-eight year old woman who was in an automobile accident twenty-seven years prior to study. (*A*) Frontal roentgenogram shows massive calcification (arrow) adjacent to the aortic knob. (*B*) Lateral esophagram shows the dense aortic aneurysm (arrow). There is also deviation of the lower esophagus owing to tortuosity of the lower portion of the descending aorta. (*C*) Left anterior oblique angiocardiogram shows the huge traumatic aneurysm at the top of the descending aorta (arrow). (*A* and *C* reproduced with permission of *The New England Journal of Medicine*.[17])

the diagnosis of *chronic* aneurysm of the aorta was made after a chance roentgenogram disclosed a mediastinal mass. In some patients, symptoms such as dyspnea, cough, and pain in the chest caused them to seek medical aid.[6,12] The characteristic site of the aneurysm at the top of the descending aorta should suggest the diagnosis. A history of chest trauma confirms it.

DIAGNOSIS AND TREATMENT

The diagnosis of *acute* traumatic aneurysm of the thoracic aorta depends upon a high index of suspicion in every accidental

injury even though there is absence of external thoracic bruises. Early, following automobile injury serial roentgenograms may reveal evidences of bleeding by progressive mediastinal widening, obscuration of the aortic arch, increased aortic density or layering due to hematoma (Fig. 2, *A–C*). This may be followed by a localized bulge at the beginning of the descending aorta just below the origin of the left subclavian artery (Fig. 2, *D* and *E*). Later, in the *chronic* phase, there is a well circumscribed mass adjacent to the aortic knob which may be pulsatile (Fig. 2, *F* and *G*). Chronic traumatic aneurysms of long standing often contain calcium deposits in the walls (Fig. 4–6). Contrast visualization of the thoracic aorta by the method of angiocardiography readily establishes the diagnosis of aneurysm (Fig. 2, *H* and *I; 4C; 5E–G* and *6C*). Other aneurysms (syphilitic, arteriosclerotic and congenital) at the top of the descending aorta need to be differentiated from the traumatic. The syphilitic are usually associated with an aortitis; the irregular, dilated and calcific ascending portion of the aorta often accompanies syphilitic aneurysms and is readily visualized by angiocardiography.[3,13] Finally, a positive serologic test for syphilis is confirmatory. Arteriosclerotic aneurysms usually have smooth, mildly dilated ascending portions of the aorta.[16] Arteriosclerotic tortuosity of the aortic knob and buckling of the brachiocephalic arteries may also simulate aneurysms.[15] Lastly, congenital anomalies of the aorta such as kinking associated with dilatation of the left subclavian artery (pseudocoarctation) may be mistaken for aneurysms. Roentgenography, especially angiocardiography, readily establishes the congenital nature of the deformity.[14] In the acute stages of aortic rupture, mediastinal hematoma may simulate mediastinal tumors or effusions.[4] Traumatic mediastinal hematomas also result from bleeding of neighboring blood vessels, especially venous channels.[10] Tumors of the mediastinum may readily be differentiated from aneurysms by angiocardiography[3] and, of course, are not associated with accidents.

In the literature there are now several cases of successful resection of chronic traumatic thoracic aorta aneurysms with replacement by homografts.[2,12,18,19] Recently, Hardin has also reported the successful resection of multiple thoracic aortic aneurysms due to trauma in a patient with replacement by an orlan graft.[6] At first, following Bahnson's technique,[1] excision of traumatic aneurysms and suture of the aorta were performed in several instances. Recurrences, however, made it necessary to reoperate and insert homografts.[2] Hypothermia, external shunting and hypotension have successfully been used as valuable adjuncts in the repair of traumatic thoracic aortic aneurysms.[6,18,19] Pleas have been made for recognition and repair of early acute aortic ruptures.[9,18] Despite these brilliant achievements there have been failures due, in part, to the resection of a chronic densely adhesive and fragile lesion, and to techniques necessary for prevention of spinal cord, lower extremity and abdominal circulatory complications. There is general agreement that resection of a traumatic thoracic aortic aneurysm is a formidable undertaking. It is for this reason that the selection of the case suitable for surgery is important.

Aneurysms that cause cough, dyspnea and pain in the back due to vertebral body pressure may be assumed to be enlarging and should be resected to relieve symptoms and prevent rupture. Enlarging aneurysms even in the absence of symptoms are due to persistent bleeding; a prime indication for surgery.

SUMMARY AND CONCLUSIONS

Acute traumatic aortic rupture associated with fractures, internal injuries and shock may be overlooked. Awareness of the role of stresses in deceleration following blunt trauma, however, should focus attention to the top of the aorta just beyond the origin of the left subclavian artery because it is the favorite site of rupture. Serial

roentgenograms of the chest for many weeks following injury permit recognition of mediastinal hematomas, hemothorax and the development of a localized bulge (aneurysm) in the region of the aortic knob. Unless routine chest roentgenograms are made after thoracic injury, many years may elapse before patients with chronic traumatic aneurysms of the thoracic aorta are diagnosed. Often, a mediastinal mass found in a routine chest roentgenogram in asymptomatic patients may be the first inkling of the aneurysm. In others, chest pain, cough, hoarseness or hemoptysis may lead to roentgen study of the thorax and discovery of a mediastinal mass and aneurysm.

Roentgenographic examination, especially roentgenoscopy and esophagraphy, are valuable for demonstrating a mass at the top of the aorta. A high incidence of calcification in the aneurysmal wall occurs in chronic cases. Angiocardiography is invaluable for diagnosis of aneurysms and readily differentiates them from mediastinal tumors. Finally, the characteristic location of traumatic aneurysms at the top of the aorta, the absence of a positive serologic test for syphilis, and the history of trauma establish the diagnosis.

Operative repair of an acute traumatic aneurysm of the thoracic aorta has not, as yet, been reported. The critical condition of the patient, often in shock and with multiple fractures and internal injuries, makes surgery hazardous and formidable. Excision of chronic traumatic aneurysms of the thoracic aorta and replacements by grafts have been successfully accomplished. However, operation should be limited to those patients who show evidence of enlargement of the aneurysm and/or develop symptoms.

Israel Steinberg, M.D.
525 East 68th Street
New York 21, New York

ADDENDUM

Since this paper was submitted for publication, the diagnosis and treatment of 6 new cases of traumatic aneurysm of the thoracic aorta have recently been reported. Eiseman, B., and Rainer, W. G. Clinical management of posttraumatic rupture of thoracic aorta. *J. Thoracic Surg.*, 1958, *35*, 347–358.

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ANALYTIC ROENTGEN KYMOGRAPHY OF THE NORMAL CARDIAC CYCLE*

By ANTONELLO LACONI, M.D., *and* GIOVANNI ZACCONE, M.D.
PALERMO, ITALY

INVESTIGATIONS on the analytic roentgen kymogram (ARK) were begun by Cignolini in 1929 with a purpose which was entirely different from that for which the kymographic methods known and employed until that time were used. His aim was to study the cardiomechanical cycle by registering the slightest possible alterations by means of suitable roentgen kymographic tracings. Cignolini introduced the original roentgen kymograph. This was improved with successive models to meet the following fundamental requirements: (1) study of the cardiac cycle at the rate of 0.01 second; (2) simultaneous registration at different points along the cardiovascular shadow; and (3) registration of several cardiac cycles on the same film.

The cardiovascular configuration was observed on a fluoroscopic screen which was provided with a graduated opaque grid. The points of diagnostic interest were marked on the graduated grid. They were then transposed to a sheet of lead with slits that was afterwards substituted for the fluoroscopic screen. The kymography was carried out by using a 30 by 40 cm. film, which moved downward at 0.02 second per 1 mm. of film. There resulted a series of tracings, side by side, that extended for 40 cm. The contacts of closure of the roentgen-ray circuit were very brief and interrupted so as to be able to obtain a transverse isochronic segment (recorded on each film). Such a transverse segment establishes the chronologic order for the comparison of the individual tracings. With the technique used in 1930–1940, there was no simultaneous visualization of the cardiovascular shadow and reference to the points of interest had to be made from the ortodiagram. Yet, Cignolini was able to establish the basic principles for rational interpretation of the normal and pathologic cardiomechanical cycle, outlining the elements of greatest value both from the clinical and physiopathologic viewpoints. In 1950, he designed a new type of apparatus called the polykymograph which, although inspired by preceding models, was entirely novel in its conception and was destined for practical clinical use. The main innovation was that the analytic roentgen kymograms (ARK) and the plane roentgen kymogram (RK) were made on the same film. To accomplish this, the total length of the ARK had been reduced from 40 cm. to 8 cm. by using mobile shutters on slits distributed on a special grid, with the adoption of many galleries and a system of rectangular shields.

This new polykymograph permits: (1) registration on the same film of the ARK and the RK; (2) lessening of the total load on the roentgen tube through a briefer period of operation; (3) reduction of the width of the slits with subsequent greater detail; (4) easy and rapid use with maximum precision of registration. The ARK can be recorded simultaneously with the ECG and other graphic representations of the cardiomechanical cycle.

A double exposure is used. The first exposure is for the analytic tracing of the cardiovascular shadow with its points of interest. The second exposure, on the same film, is added for visualization of the alterations in the plane RK, as demonstrated with a mobile or fixed grid. The ARK is exposed at a focus distance of 90 cm. and the plane RK at a focus distance of 150 cm. This last distance was chosen to enable the simultaneous evaluation of cardiac volumetrics. The reading of the ARK is done from the bottom up, since the analytic tracings are always registered with a fixed

* From the Institute of Medical Radiology and Physical Therapy of the University of Palermo (Italy). Director: Prof. P. Cignolini.

grid. The plane RK is read, as usual, by following the standard procedures. The effect on the roentgenographic appearance due to respiration must also be taken into consideration. Cignolini, in studying this

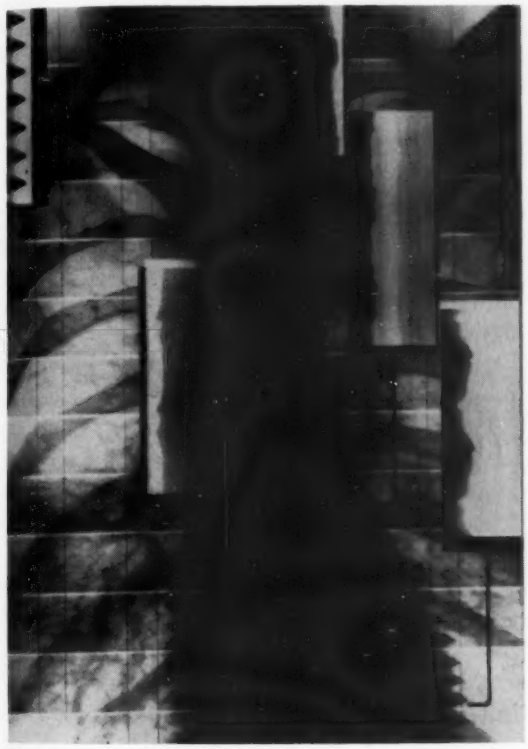

Fig. 1. Polykymogram of normal subject. Velocity 1 mm. per 0.03 second. Analytic roentgen kymography (ARK) related to surface roentgen kymography (RK) (12 mm. mobile grid). ARK of inferior portion of left ventricle, left auricle, aorta, and right inferior arc. The right horizontal lines at the lower end of the vertical lines indicate the points of origin of the ARK. The tracings are displaced 5 cm. at the top to avoid superimposition on the cardiovascular shadow, which would prevent morphologic and volumetric evaluation. There is a chronographic tracing at the upper left.

aspect, noted that the cardiac cycle, as expressed by ARK, does not undergo substantial modification during the various phases of respiration.

The elements that constitute the roentgenographic document obtained by the polykymograph (Fig. 1) are: (a) the cardiovascular configuration obtained by the plane RK with the fixed or mobile grid; (b) the ARK registered at points of maximum interest for diagnostic purposes. As already stated, each tracing of the ARK is 8 cm. long. From the bottom of each tracing, there descends a vertical line of 5 cm. length, with a horizontal pointer to the landmark of interest of the cardiovascular shadow to which the tracing refers. The upward displacement on the film of the group of analytic tracings is done with a technical system, the purpose of which is to avoid the overlapping of the ARK with the cardiovascular shadow. This permits an optimum evaluation of the configuration of the cardiac shadow; (c) the chronographic registration (as shown at the top on the left) of the even movement of the film displacement during the ARK exposure. The speed corresponds to 0.02–0.03 second per 1 mm. of film; (d) the simultaneous registration on the same film, through the use of an oscillating ECG pen in front of a slit of the analytic grid, of the ECG, the phlebogram and other studies of the cardiac cycle.

The normal pattern of the ARK was described by Cignolini in 1934[1] and was presented again in a recent text book.[2] Slight variations may be found due to individual and constitutional factors, depending on whether the kymographic tracings were made in a cephalad or caudad direction.

THE NORMAL ARK OF THE LEFT VENTRICLE

The normal ARK of the left ventricle reflects the changes in the volume of the heart. Some of these are due to anatomic position. The right ventricle, for example, which rests with a large base on the diaphragm, can produce an increase in width of certain excursions caused by the displacement transmitted to the left ventricle. The ventricular kymogram is of cardinal importance in cardiac mechanics both because it furnishes useful morphologic data for the interpretation of normal and pathologic states and because it clearly indicates the beginning of the mechanical ventricular systole (Fig. 2*c*).

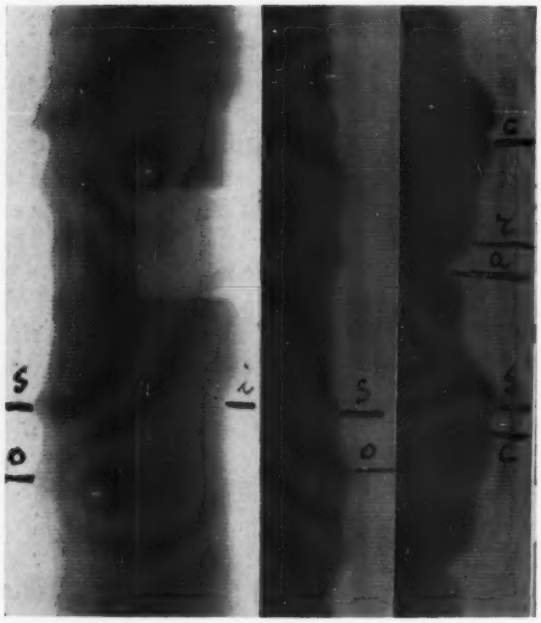

Fig. 2. Reproductions of the four analytic roentgen kymograms (ARK) traced on the film in Figure 1. The ARK tracings are placed side by side in similar chronologic arrangement. *Outer left:* Right inferior arc. *Inner left:* Aorta. *Inner right:* Left atrium. *Outer right:* Inferior portion of left ventricle. In the left ventricle the two phases of the diastolic filling are well distinguished (*a-r* and *r-c*). The same can be said of the precise beginning of ventricular systole (*c*) and the phase of systolic output (*s*). The atrial systole (*o*) is practically simultaneous in both atria. The chronomorphologic elements of the ARK of the right inferior arc are prevalently of the atrial type: slight presystolic atriodiastasis followed by atrial systole (*o*), which is brief and slightly indented, and by the cusp *s*, due to transmission to the atrium from the ventricles by raising of the atrioventricular plane. Note the paracardial vessels that flank the ventricular ARK.

This beginning is an incident which causes a vigorous effect on the entire left ventricle and constitutes a sure reference point for the chronologic localization of the atrial and vascular phenomena. It is demonstrated very accurately in the ARK. There are some points indirectly represented by the sphygmic foot of the large vessels, which are also chronologically more exact than in the sphygmograms. The commencement of systolic contraction in ARK of the left ventricle is identified as a protosystole of point *c*, affecting constantly and simultaneously all points of interest of the ventricle. On the electrokymogram (EKY) the chronologic individualization of such a phase has always constituted an important problem that should have its logic solution in the simultaneous use of at least two photocells, one situated on the left ventricle and the other on the arc of a major interest for the analysis. Actually, diverse factors prevent on the EKY the localization of such a precise point as the beginning of the ventricular contraction. Above all, the EKY tracing is the result of an uncontrollable combination of changes in position of the cardiac margins and the variation of positions and density of the paracardial shadows. The blood vessels, bronchi and pulmonary alveoli reflect rhythmically the variations in volume and position of the adjacent sections of the heart, resulting in a complicated nonsynchronized visualization of the paracardial and cardiovascular structures. This fact is even more apparent in pathologic conditions where the paracardial shadows are more dense and show a more accentuated movement, rendering the chronology and exact evaluation of the relative changes in size of primary importance.

There need to be considered: (a) the unequal sensitivity of the central and peripheral surfaces of the photocell; (b) the inverse relationship between frequency of registration and width of excursion, according to which the width of excursion diminishes as the frequency of the light signal increases; (c) the impossibility to establish a proportional relationship between the movements of the cardiovascular margins and the corresponding ECG waves; therefore, ruling out a means of measuring.

The photoelectric receptor registers the movements insofar as these determine variations in the illumination of the slits placed on the margins of the cardiovascular shadow and depending on the size of the subject, and on the voltage and amperage, although independent of the variations of the potentiometer. An important complicating factor is the delay in the transcrip-

tion due to the system of filters which varies from machine to machine and which must be known everytime a coordination with other clinical graphs is desired. This disadvantage leads to inaccurate morphologic evaluation and uncertainty of the stable points of reference—a fact which renders necessary the use of time pacers of different types to obtain the chronologic localization. The ECG is often employed in this work even though the relationship between the mechanical and electrical waves is not constant and there exist individual variations.

As is known, the ECG registers solely phenomena of systolic depolarization and repolarization and cannot be used for the localization of the phases of the cardiac cycle. Even with the phonocardiograph, the determination of the beginning of the ventricular mechanical cycle is not to be considered dependable. It is difficult, in fact, to recognize on the first tone, the first oscillations produced by the heart contraction. Likewise, the carotid sphygmography, which has often been used for this purpose as a pacemaker of pneumographic procedures, has an inherent delay. The ARK is the only method available which registers directly or indirectly with great chronometric accuracy the beginning of the ventricular systole.

The impossibility of a chronometric localization of the beginning of the ventricular systole by EKY has introduced a fundamental error in the interpretation of the pathologic heart, leading to frequent contradictions.

The principal features of the ventricular ARK cycle may be summarized as follows: From the beginning of the diastolic filling there is a definite angle between the preceding phase of minimum volume and the abrupt elevation of rapid filling. The apex of this angle *a* represents the opening of the cuspid valve. From *a* there is a sharp slope, corresponding to about one-half of the ventricular lateral excursion, called diastolic rapid filling, *a-r*. Between the succeeding angle *r* and the beginning of the

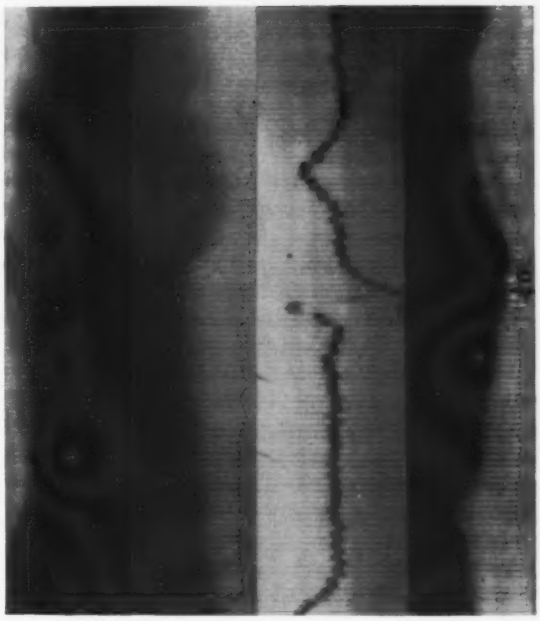

Fig. 3. Simultaneous registration of the ARK and ECG in right II lead, with an oscillating pen in front of one of the slits of the analytic grid. This permits an accurate comparative study of the electric and mechanical elements of the cardiac cycle.

ventricular systole *c*, there is a slow ventricular filling, *r-c*. In this phase the increase in volume is much less and the lateral excursion is gradual. The apex from the diastolic side and the rapid centripetal slope which follows indicate the beginning of the systole *c*. This point, as has been said, is rigorously simultaneous on the ARK, taken from the various sections of the left ventricle. The brief segment that runs between the *c* and the apex of the succeeding U-shaped curve represents the phase of isometric systole. With the beginning of the systolic outflow, the U pattern is inverted with the formation of the sharp cuspidated or rounded off positive wave *s*, which is the result of tone, thickness and elasticity of the heart and, in certain conditions, the displacement due to right ventricular hyperkinesia. From the bottom of the U curve to point *s*, there is the period of rapid ventricular ejection. From the apex *s* the slope descends more or less rapidly, until the minimum volume is reached. The systolic centripetal slope is followed by the

rapid filling during the following cycle but more frequently between the systolic descent and the succeeding diastolic elevation there is noted a baseline of minimum volume pertaining to the telesystole and isometric diastole. In this baseplane there is often a dome-shaped wave (*i*) which in experimental conditions has been recognized as being chiefly of positional origin.

THE NORMAL ARK OF THE RIGHT ATRIUM

The ARK of the right inferior arc (Fig. 2) is often of a mixed atrioventricular type due to the fact that the thin atrial wall almost covers the right ventricle, the activity of which is thus transmitted to the right atrium. On the kymogram of the right atrium, the atrial systole (*o*) is always clearly shown. Often this is the only sign of atrial activity, the remainder of the tracing reflecting the transmitted effects of the ventricular activity, as, for example, the wave of systolic outflow (*s*) and, less frequently, the beginning of ventricular systole (*c*). During the ventricular systole, the atrial ARK shows a downward slope followed by gradual diastasis due to the continuous flow of venous blood during the last part of the systole. This is followed by a tumble corresponding to the opening of the atrioventricular valve and finally by the presystolic repletion until the succeeding atrial systole (*o*). Therefore, although the normal ARK of the right atrium may have a constant shape, its character varies according to the dominating elements transmitted from the ventricle. The effects of transmission are more accentuated in the caudal points of the right inferior arc.

THE NORMAL ARTERIAL KYMOGRAM

The ARK of the aorta and pulmonary artery (Fig. 2) shows the following characteristics: There is early appearance of the sphygmic wave in the pulmonary artery; in the aorta, the systolic wave produces a semicircular incisure (*i*) and there is a uniform diastolic phase. On the ARK of the aorta, the ascending phase is brief, followed by a long descending diastolic phase interrupted by the incisure, which is less pronounced than in the pulmonary artery. The width of excursion is small in both vessels, being about 2 mm. on the average.

CHRONOLOGIC RELATIONSHIP BETWEEN ARK AND ECG

In the ECG the Q deflection precedes the beginning of the mechanical systole (*c* on the ARK of the left ventricle) by about 0.04 seconds. In most cases the *c* coincides with the apex of the R deflection of the ECG. This chronologic relationship has been found to be constant in all tracings made simulanteously on points cephalad and caudad to the left ventricle. In the right ventricle, the beginning of the systole can be seen 0.02–0.04 second before the systole of the left ventricle. Study of the chronologic relationship between ARK and ECG of the aorta and pulmonary artery shows that the commencement of the sphygmic wave in the aorta appears about 0.1 second after the Q deflection in the ECG, while the corresponding wave in the pulmonary artery is delayed to only 0.03–0.06 second after the Q deflection. The P deflection of the ECG precedes by 0.03–0.06 second the mechanical atrial systole (*o*). Sometimes it is possible to note a slightly earlier appearance of the right atrial systole than of the corresponding left atrial systole.

SUMMARY

An analytic roentgen kymographic method is described and the normal ventricular, atrial, and vascular patterns are discussed.

Antonello Laconi, M.D.
Istituto di Radiologia
Della Università di Palermo
Palermo, Italy

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CONDITIONS PERMITTING REGURGITATION OF RADIOPAQUE CONTRAST MATERIAL THROUGH THE INTACT MITRAL VALVE*

By HERBERT M. STAUFFER, M.D., *and* MORTON J. OPPENHEIMER, M.D.
PHILADELPHIA, PENNSYLVANIA

REGURGITATION of radiopaque contrast material through normal mitral valves has been demonstrated in dogs by an image intensifier cineroentgenographic technique.[7] Such regurgitation was observed in situations during which the phase of diastasis of the cardiac cycle is increased.[4] These conditions include marked bradycardia, sinus arrhythmia, temporary asystole, and compensatory pause after a premature contraction.[5] This regurgitation through a normal valve during vagal asystole has also been confirmed by electrokymography.[3]

Since the arrhythmias enumerated above are common during clinical left ventriculography,[1] the possibility of mitral regurgitation occurring on this basis becomes an important consideration in interpreting such a diagnostic study. Previous studies in dogs by others[2, 8] using conventional roentgenography have not shown regurgitation through intact mitral valves presumably because significant arrhythmias were not encountered during contrast material injection.

The present study was performed to demonstrate regurgitation through a normal mitral valve using conventional roentgenographic techniques.

METHOD

Dogs were anesthetized with pentobarbital sodium, 35 mg./kg. The left vagus nerve was exposed in the neck. The peripheral end of the divided nerve trunk was stimulated electrically. During this vagal stimulation and for a short control period just prior to it, roentgenograms were exposed at intervals of 0.7 second. At the time of cardiac inhibition (asystole), an opaque medium (10 cc. 90 per cent miokon) was injected into the left ventricle by way of a catheter introduced via the carotid artery and aorta. The time of roentgenographic exposures was indicated automatically on the electrocardiographic paper. The dogs were in supine decubitus position during the experiments.

RESULTS

Roentgenograms (Fig. 1) were obtained which demonstrated regurgitation of the radiopaque contrast material from the left ventricle into the left atrium during vagal asystole. The regurgitation was diastolic in time. The degree of regurgitation was graded according to Paul[4] and indicated on the electrocardiographic record (Fig. 2). In the case illustrated two exposures demonstrated regurgitation which filled the pulmonary veins (Grade IV, Paul).

CONCLUSIONS

Regurgitation through a normal mitral valve may be demonstrated by conventional roentgenographic techniques during vagal asystole (long diastasis).

Possible causes of "physiologic regurgitation," *i.e.*, long diastasis, must be considered in evaluating the clinical left ventricular angiocardiogram. Simultaneous electrocardiographic records correlated with the roentgen exposures are essential. Significant organic regurgitation should be demonstrable with each systole throughout the period of injection.

These conclusions are borne out by the

*From the Departments of Radiology and Physiology, Temple University Hospital and School of Medicine, Philadelphia, Pennsylvania.
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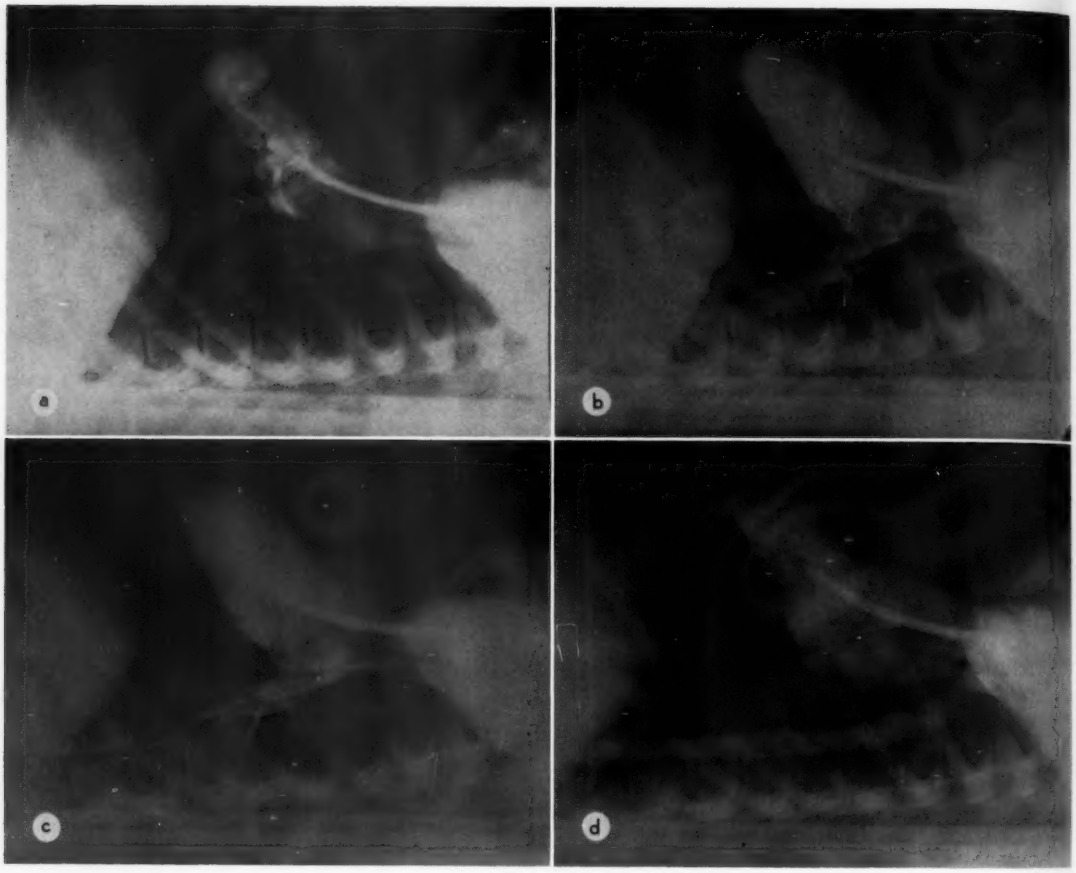

Fig. 1. Mitral regurgitation of opaque contrast material during vagal asystole; injection into left ventricle via aortic catheter. The timing of the numbered exposures is indicated on the top line of electrocardiographic record (Fig. 2). (*a*) Exposure #78. Minimal regurgitation (Grade I). (*b*) Exposure #80. Marked regurgitation (Grade IV). (*c*) Exposure #81. Continued regurgitation extending into pulmonary veins (Grade IV). (*d*) Exposure #82. Clearing of contrast material from atrium with resumption of cardiac contractions after end of vagal stimulation (Grade I).

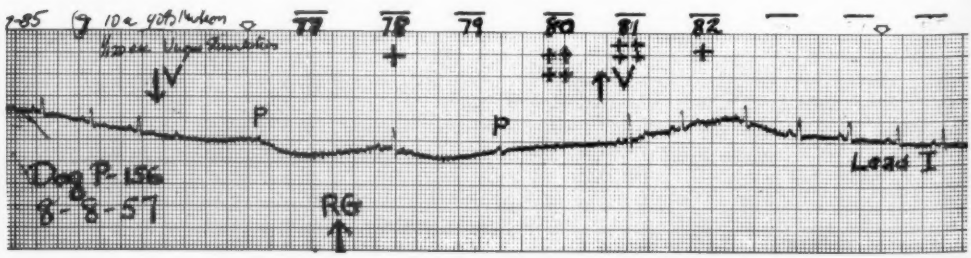

Fig. 2. Electrocardiogram. Serial roentgen exposures top line; duration $\frac{1}{2}$ second, interval 0.7 second. ↓V = onset and ↑V = end of vagal stimulation. P = P waves of ECG. RG = start of contrast material injection. Plus marks under roentgen exposure numbers = degree of regurgitation according to Paul (4+ or Grade IV = regurgitation which fills pulmonary veins).

observations of Ruhde[6] based on his experience in clinical angiocardiography in Stockholm, Sweden.

Herbert M. Stauffer, M.D.
Temple University Hospital and
School of Medicine
Philadelphia 40, Pennsylvania

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LUNG CHANGES IN ACQUIRED HEART DISEASE*

By PETER KERLEY, M.D., F.R.C.P., F.F.R.
LONDON, ENGLAND

CARDIOVASCULAR roentgenology for most of its history has been a study of heart size and of selective chamber enlargement. Though many of us, even in the early days, were interested in associated lung findings, we could not get very far because of low power units and broad focus tubes giving poor detail. The spectacular results of cardiac catheterization, angiocardiography and surgery have stimulated interest in this aspect and there is little doubt that the straight roentgenograms in many diseases are a faithful reproduction of the altered hemodynamics. They very occasionally yield more information than the catheter or the angiogram, since the straight roentgenograms are usually taken in an ambulant out-patient, while the catheter and contrast studies are made in a hospitalized patient who has been sedated.

In acquired heart disease, the lung changes are of most value in mitral valve lesions and left heart failure.

In mitral valve lesions, the cardiologist can usually say with confidence that the patient has predominant stenosis or incompetence, but in between there are many cases where it is impossible to determine which condition is dominant and in this group roentgenology is of greatest value. The size of the ventricles, the size of the left auricle and valvular calcification, which were considered sound guides as little as five years ago, are now known to be fallacious in many instances.

There are six roentgenologic features which appear in the lungs in mitral valve disease. Three of these are primary and three secondary. The primary ones are (1) distention of veins, (2) distention of lymphatics, and (3) distention and contraction of arteries. The secondary changes are (4) hemosiderosis, (5) bone formation in the lungs, and (6) pulmonary fibrosis, secondary emphysema and atelectasis. Obviously, not all are present in any given case but combinations of them are, and when correlated can be decisive in establishing the pressure group.

In the normal roentgenogram of the lungs, the upper lobe veins are either invisible or barely visible while the lower lobe veins, especially on the right, are quite easy to identify. When the mitral valve area becomes narrowed, the raised left atrial pressure is at once transmitted to the pulmonary veins and the upper lobe veins stand out clearly. The veins, which are wider than the arteries, run for several inches without bifurcating and usually only one bifurcation is visible on straight roentgenograms. They are rather like staghorns extending up from the hili. The base of the vein in the hilus is broad and twice the width of the corresponding artery. This distention of the upper lobe veins occurs early and may be seen shortly after the phase of acute carditis when there is no clinical evidence of a valvular lesion. This early upper lobe venous distention is probably an indication of a generalized carditis. If the disease results in fibrous ankylosis of the valves and mitral incompetence, the distended upper lobe veins will gradually disappear unless left ventricular weakness comes into play.

However, if mitral stenosis is established, the upper lobe veins remain distended and paradoxically the lower lobe veins become invisible. This is a dramatic roentgenologic feature of mitral stenosis. I had noted it in a lecture to the Faculty of Radiologists some years ago but misinterpreted its significance. Recently Simon[4] explains this paradox as follows. In the erect position the venous pressure is higher in the lower lobes, so when there is mitral obstruction, venous vasoconstriction is greater in the lower

* Presented at the Fifty-eighth Annual Meeting of the American Roentgen Ray Society, Washington, D. C., October 1–4, 1957.

lobes with a corresponding rise in the upper lobe venous pressure. The cardiac output is maintained by an increased flow through the upper lobes due to the basal vasoconstriction; hence the upper lobe venous distention is only indirectly due to the raised atrial pressure. (This hypothesis would also explain the greater frequency of infarcts, infection and atelectasis in the lower lobe, which I will discuss later.) In the most advanced cases when pulmonary hypertension is severe, the circulatory volume even through the upper lobes is diminished and all venous shadows tend to disappear.

Once the venous pressure rises above 25 mm. of Hg (the value of the plasma osmotic pressure), there is increased transudation of fluid and the pulmonary lymphatics become visible. The intercommunicating lymphatics, seen as the transverse lines at the bases, are the first to appear, but if an acute episode occurs the deep lymphatics also show up. The latter have a bizarre appearance, being about 1 mm. thick and running as long straight lines in an apparently haphazard fashion. The transverse basal lines are seldom more than 1 inch long while the deep lymphatic lines vary between 2 and 5 inches in length. For reasons unknown, the engorgement of the deep lymphatics is usually transient while the intercommunicating ones vary little, except with prolonged rest in bed, when they may become less conspicuous or disappear.

Many of us over the years had correctly inferred the nature of these lines, but it was only recently that I obtained anatomic proof of their nature from injection of the subpleural lymphatics in a patient who died suddenly during a valvotomy. Since this paper was prepared a number of papers have appeared proving from anatomic specimens that the transverse lines are due to dilated lymphatics in the interlobular septa. Similar linear or interstitial edema can occur in acute or chronic left ventricular failure and in some forms of malignant disease and pneumoconiosis but never to quite the same extent as in mitral disease. In acute left ventricular failure the whole

FIG. 1. Venous distention in mitral valve disease. The right lower lobe vein is clearly visible. (Pressure group 1.)

pulmonary lymphatic system may be distended for a short time and lines, (a) the superficial lymphatics, (b) the intermediary lymphatics, and (c) the deep lymphatics, can be seen for twenty-four to forty-eight hours, unless the patient recovers or dies. In chronic left ventricular failure only a few transverse lymphatics are visible and the others are largely overshadowed by venous

FIG. 2. Laminagram showing distended upper lobe veins and contracted lower lobe veins in mitral stenosis.

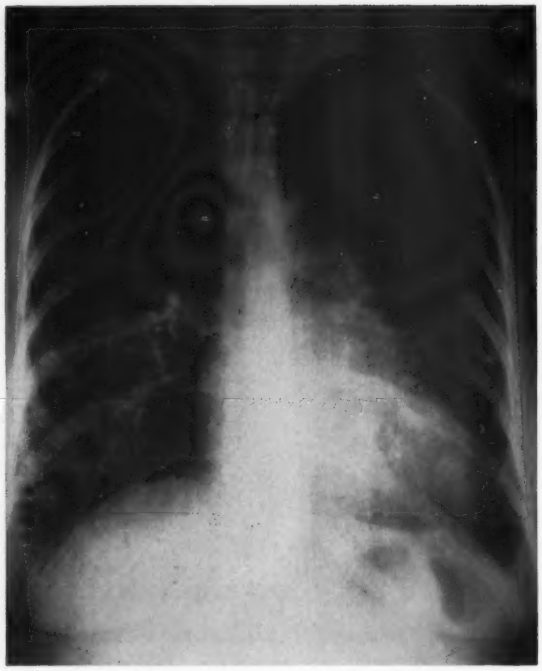

Fig. 3. Distention of the intercommunicating lymphatics with contraction of the pulmonary arteries.

engorgement in all lobes and the hili. In mitral stenosis the transverse lines are more extensive and persistent. It is reasonably certain that these lines are a true reflection of raised pulmonary intravenous pressure. Some authors have inferred that they are also an indication of raised pulmonary artery pressure. This cannot be accepted unless the high arterial pressure is secondary to the high venous pressure. In pulmonary hypertension due to causes other than mitral disease, the lines do not appear. In mitral disease the lines alone should never be accepted as an indication of pulmonary artery hypertension.

The pulmonary artery changes are easier to recognize. In minor degrees of mitral stenosis (functional group 1) there is little or no alteration. Slight enlargement occurs but is too difficult to estimate. In severe degrees of pulmonary hypertension the main stem, the two main branches and the second degree branches are enlarged while the peripheral branches are narrowed and tortuous. These changes are gradual, and Goodwin and others[2] have graded them by angiocardiography. It is possible to correlate the angiographic grading on the straight roentgenograms and indeed Fleischner and

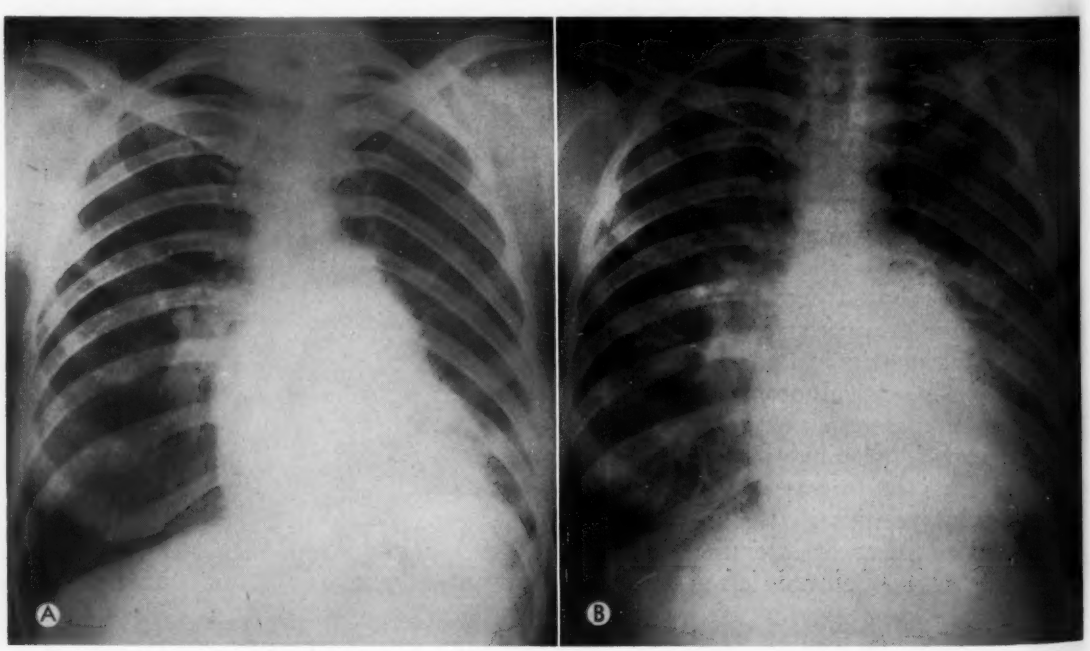

Fig. 4. (*A*) Thrombosis of the right lower lobe artery in mitral stenosis. (*B*) Same case as *A* during an attack of acute dyspnea. The deep lymphatics are distended.

Sagall[1] had already done so. It can be done more easily by laminagraphy. I have repeatedly noted that vascular contraction in primary and secondary pulmonary hypertension first manifests itself roentgenologically in the left upper lobe. A possible explanation for this hypertranslucency of the left upper lobe is that a distended main pulmonary artery stem can squeeze the left upper lobe vein.

It is only in the severe degrees of pulmonary hypertension that these signs are visible, and simultaneously the engorged veins and the interstitial lymphatic lines tend to become less obvious as the functional vasoconstriction becomes organic and the cardiac output is reduced. In a gross case, and particularly in young pregnant women, we may even see a relatively small heart with moderate enlargement of the left auricle and pulmonary artery and hypertranslucent lungs, and the patient is at death's door. The hypertranslucency is overlooked and regarded as technical when in fact it means contraction of all the vessels.

Fig. 5. Mitral stenosis with severe pulmonary artery hypertension. Note the contraction and tortuosity of the right lower lobe vessels. There is associated lymphatic distention.

Fig. 6. Extensive bone formation in mitral stenosis. An aortic leak was present but not dominant.

There is one other condition which must be considered as a cause of hypertranslucency in association with mitral disease. In the last two years I have seen 3 cases with pulmonary stenosis coupled with mitral stenosis. Mild degrees of pulmonary stenosis are very common, perhaps more common than atrial septal defect and, of course, it predisposes to rheumatic disease. The differential diagnosis is not too difficult. If mitral stenosis is coupled with pulmonary valve stenosis, the main stem is enormous and the right main branch very small. If stenosis is infundibular, the main stem is small and the main branches very small. In the presence of a big left auricle and a small or normal right auricle, the discrepancy between the arteries is an obvious clue to a double lesion.

The significance of these signs can be summarized as follows: distended upper lobe veins, contracted lower lobe veins and marked interstitial lines mean that mitral stenosis is dominant over mitral incompetence; distended upper lobe veins plus interstitial lines and small peripheral arteries indicate predominant mitral stenosis with pulmonary hypertension; dis-

Fig. 7. Honeycomb fibrosis in the right lower lobe in chronic mitral valve disease.

tended upper and lower lobe veins indicate a low pulmonary pressure, so that whenever the lower lobe veins are visible, it is safe to assume a low pressure. Simon in Guy's hospital is going much further than this and he grades the mitral lesion into six groups on these patterns.

Hemosiderosis is fairly common in mitral stenosis. It appears as a fine granular pattern like a first stage pneumoconiosis or as dense larger miliary opacities 1 or 2 mm. in diameter. In the early years of this century when rheumatic heart disease was more prevalent, physicians used to search for hemosiderin in the sputum as a sign of mitral disease. I do not know its significance. Goodwin and his co-workers[9] believe that it is an indication of pulmonary artery hypertension and a certain one if coupled with transverse interstitial lines. I think that the number of cases in which it has been correlated with hypertension is too small to justify this conclusion. Physiologically, hemosiderin should only be deposited when the pH of the lungs is altered by severe venous congestion and it can disappear after valvotomy.

Bone formation in the lungs in heart disease is almost conclusive of mitral stenosis. The pathologic process has not been explained, but it is certainly not a sequel to a rheumatic pneumonia as some earlier workers thought. It occurs in about 3–5 per cent of mitral stenotics. I have never seen it in dominant mitral incompetence or aortic incompetence or aortic stenosis of rheumatic origin and the only other disease in which I have seen it develop was in a hyperkinetic state due to a gross arteriovenous aneurysm from a gunshot wound in the first world war. It is quite easy to identify in a severe case. There are multiple dense opacities like calcified tubercles or histoplasmic granulomas scattered rather widely at both bases. Unlike tubercles and histoplasmic nodules, they are not clumped together and they vary in shape. Some are round but many are angular or oval. In the early stages they are concealed by the breast shadows and often misinterpreted as distended end-on vessels. Exceptionally, they develop in the upper

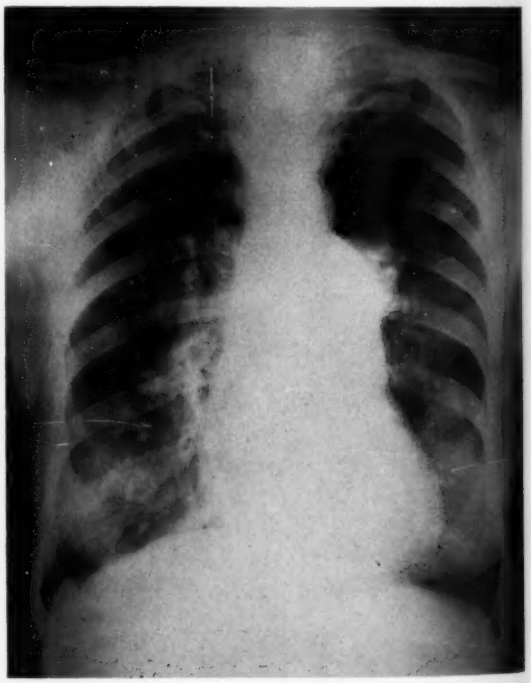

Fig. 8. Hypertranslucency of the left upper lobe (?) due to compression of the upper lobe vein by the distended pulmonary artery. Such localized hypertranslucency is common in mitral disease.

lobes. Obviously, this ossification is a result of the altered hemodynamics, and its selectivity for the lower lobes connects it with spasm of the lower lobe veins. I have never seen it without associated interstitial lines. It is a diagnostic sign of mitral stenosis, but I have been unable to correlate it with the pulmonary artery pressure.

Little attention has been paid to the grosser lung changes. We often see a severe mitral lesion with splaying and compression of the bronchi, thickened diaphragmatic pleura, fibrous tissue strands and honeycomb areas, hypertranslucency due to overdistention and occasionally lobar and segmental atelectasis. These changes are invariably localized to the lower lobes. The diagnosis of mitral disease is obvious and one almost automatically concludes that the patient has had previous pulmonary infarcts or attacks of failure or recurrent infections. As often as not there is no history of previous infarction or failure and occasionally there is only a history of a minor pulmonary infection. At autopsy the grossest fibrotic changes are discovered. Silent infarcts will account for some of the pathology, and chronic edema inevitably results in failure, but since the changes are always in the lower lobes, they must also be related to venous spasm or thrombosis and indirectly to spasm or thrombosis of the bronchial arteries. When the nutrition of the lung is thus disturbed, compression of the bronchi by a big left auricle easily causes atelectasis. Pressure atelectasis is only a minor factor although it may dominate the clinical and roentgenologic findings.

Fig. 9. Thrombosis of a right lower lobe vein in mitral stenosis. Note the abrupt termination of the vein and its course into the auricle at right angles to the artery.

Fig. 10. Hypertensive failure. This is a very obvious case. Note the ill-defined hazy hili and the small bilateral effusions, lamellar on the left. There are some transverse lines on the left.

There are many patients deemed inoperable because of chronic failure and chronic basal infection and collapse. Many of these are embarrassed more by their secondary pulmonary pathology than by their mitral deformity. In most instances these gross pulmonary changes are more pronounced in the right lower lobe although the left lower lobe by no means escapes completely. I think that some of these in-

operable cases might well become operable by a preliminary lower lobectomy or at least their lives could be prolonged and made more tolerable by removal of an added cardiac embarrassment. This is theoretic but it is certain that many of these people die a pulmonary and not a cardiac death. Such complicated and apparently hopeless cases are a challenge as much to radiology as to surgery.

Exceptional complications of mitral stenosis are pulmonary artery thrombosis and pulmonary vein thrombosis. Thrombosis of a main artery can occur gradually. It results in gross hypertranslucency of the affected lung and, being unilateral or unilobar, is easily distinguished from hypertension.

Pulmonary venous thrombosis may occur as a direct extension of a thrombus in the left auricle, or as a sequel to the combination of pulmonary artery contraction, venous spasm and chronic edema. The veins are the anchors of the auricle and their position in the lungs determines the direction in which the auricle will enlarge. Anatomically it is quite possible for an auricle enlarging upwards and to the left to kink the right lower lobe veins and vice versa. The importance of venous thrombosis in surgical assessment needs no elaboration. It could easily be demonstrated by angiocardiography, a rather hazardous procedure in mitral disease. I think it can be demonstrated on straight roentgenograms if there is no effusion. A thrombosed right lower lobe vein is rigid, narrow (1–3 mm.) and denser than an artery and runs into the heart shadow at a right angle to the artery. If a thrombosed vein is directly visible or suspected because of localized hypertranslucency and increased interstitial lines in one area, it should be possible to reveal the pathology clearly by good laminagraphy.

The successes of surgery in valvular and congenital abnormalities have made us concentrate on these perhaps at the expense of the more common hypertensive and ischemic left heart lesions. In Great Britain and probably in the United States there are many patients with left ventricular failure masquerading as bronchitics and asthmatics. In addition, there are many patients with trivial dyspnea, indefinite pain and equivocal electrocardiographic changes which baffle the cardiologist. The obvious roentgenologic signs of left ventricular failure are distended upper lobe veins, venous swelling and edematous glands making the hilar shadows big and ill-defined, slight interstitial edema with transverse lines at the bases and occasionally lamellar pleural effusions. These signs are conspicuous by their absence in bronchitis or asthma.

In a certain percentage of cases of left ventricular failure, some combination of these signs appears before there is any clinical evidence of failure. Short found them positive in 43 out of 51 patients with left ventricular failure or left ventricular enlargement. In 12 out of 17 cases where all clinical findings by experienced cardiologists were doubtful, the roentgenologic signs were correct and failure occurred a few months later. If a hypertensive patient with minor dyspnea or even without symptoms has interstitial lines and prominent left upper lobe veins, he is on the verge of failure and this also applies in patients with doubtful cardiac pain and a normal sized heart. If there is also a thin lamellar effusion, impending failure can be diagnosed with confidence. It is not within the scope of this paper to elaborate these findings, but there is no doubt about their validity and the diagnosis is as simple as that of a duodenal ulcer. It is quite easy to learn by studying serial roentgenograms of a patient responding to treatment for severe failure. The minimal changes, as the gross pulmonary edema and congestion fade, are identical with those in the prefailure stage.

There are other conditions which could be included under my title, notably the thrombo-embolic lungs, the varying edemas and the collagenous diseases. The appearances in mitral disease and left heart failure

are only the beginning, and the key to many unsolved problems lies in straight roentgenography and a knowledge of the anatomy and physiology of the vascular bed of the lungs.

Westminster Hospital
and National Heart Hospital
10, Upper Wimpole Street, W.1.
London, England

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DISEASES OF THE HEART AND GREAT VESSELS: THORACIC ROENTGENOGRAPHIC MANIFESTATIONS

By ANDRÉ J. BRUWER, M.B., CH.B.*
Section of Roentgenology
and
CORRIN H. HODGSON, M.D.
and
JOHN A. CALLAHAN, M.D.
Section of Medicine
Mayo Clinic and Mayo Foundation†
ROCHESTER, MINNESOTA

CONGENITAL and acquired malformations of the heart and the major vessels arising from it frequently have roentgenographic manifestations in the thorax that mimic those of primary pulmonary or mediastinal diseases. An acquaintance with these manifestations may, on occasion, save one considerable embarrassment. Furthermore, several of these conditions are sufficiently fascinating to make it difficult to resist reporting them. Over the past several years at the Mayo Clinic, we have been interested in this type of case and have made a collection which we think may be of interest to others. We also hope that presenting them in a coordinated group may assist in stimulating a greater awareness of their occurrence.

The manifestations which we will present depend, for the most part, on the collection of fluid transudates in certain parts of the thorax or on the alteration of the mediastinal shadow by anomalies of development or acquired alterations of the cardiovascular structures.

We may conveniently and arbitrarily classify the roentgenographic entities to be discussed as follows:

I. Pulmonary entities
  A. Vascular congestion in cardiac failure
  B. Vascular congestion in left-to-right shunts
  C. Lymphatic congestion
  D. Pulmonary hemosiderosis
  E. Pulmonary ossification in mitral stenosis
  F. Pulmonary embolism and infarction
  G. Acute pulmonary edema
  H. Decreased pulmonary vascularity
  I. Enlarged bronchial arteries
  J. Anomalous venous connection of the right lung
  K. Intralobar bronchopulmonary sequestration
  L. Pulmonary arteriovenous fistula
II. Pleural entities
  A. Common basilar effusions
  B. Uncommon pleural effusions
    1. Loculated interlobar effusions
    2. Mediastinal pleural effusions
    3. Subpulmonary pleural effusions
III. Entities of bony thorax
  A. Coarctation
  B. Intercostal arteriovenous malformation
  C. Aneurysms
IV. Mediastinal manifestations of cardiovascular disease
  A. Conditions affecting the aorta and its brachiocephalic branches
    1. Coarctation of aorta
    2. Kinking of aortic arch
    3. Right-sided aortic arch
    4. Dextroposition of the descending thoracic aorta
    5. Aneurysm of sinus of Valsalva
    6. Aneurysms of the thoracic aorta
      a. Traumatic
      b. Congenital
      c. Acquired aneurysms—atheromatous and syphilitic
    7. Aneurysms and buckling of innominate and carotid arteries
    8. Anomalous right subclavian artery and dysphagia lusoria

* Now at Tucson Medical Center, Tucson, Arizona.
† The Mayo Foundation, Rochester, Minnesota, is a part of the Graduate School of the University of Minnesota.

9. Compression of the esophagus by descending thoracic aorta

B. Conditions affecting the pulmonary artery
   1. Pulmonic stenosis (without interventricular septal defect)
   2. Absence of a pulmonary artery
   3. Aneurysm of the pulmonary artery
   4. Anomalous course of left pulmonary artery to right of trachea
   5. Chronic massive thrombosis of pulmonary artery

C. Conditions affecting the thoracic veins
   1. Anomalous venous connection of the right lung
   2. Total anomalous pulmonary venous connection
   3. Superior vena cava
   4. The azygos vein

D. Conditions affecting the heart and pericardium
   1. Kartagener's triad — dextrocardia, bronchiectasis and sinusitis
   2. Calcified myocardial infarcts
   3. Ventricular aneurysm
   4. Pericardial cysts and diverticula

No attempt will be made at exhaustive discussion of any of these conditions. When possible, one or two illustrative roentgenograms will be presented and references will be given to pertinent articles that deal more fully with a particular aspect.

PULMONARY ENTITIES

Vascular Congestion in Cardiac Failure. It is not our intention to discuss the more subtle differences in pulmonary vascular congestion as seen in left ventricular and right ventricular failure.[63,64] Rather do we refer to those cases of myocardial failure in which gross congestive changes occur in the lungs.

The roentgenographic changes of pulmonary congestion of cardiac failure may include the following findings:[33] generalized accentuation of normal pulmonary vascular markings accompanied by an amorphous haziness of the over-all picture; basal edema which tends to obscure or obliterate the diaphragmatic outline and the costophrenic sulci; pleural effusion which is more common in left than in right heart failure;[64] scattered areas of irregular consolidation. These findings differ considerably from those of the so-called batswing type of pulmonary edema which will be referred to later.

Thus, for instance, Figure 1*a* represents a case in which it would not be unreasonable to include a roentgenologic diagnosis of basilar bronchiectasis in the differential diagnosis. Note, however, that the heart is enlarged, and that, on medical treatment of the cardiac condition, the lungs have become normally aerated (Fig. 1*b*). It should be noted that in mitral stenosis the pulmonary changes are progressive, whereas in left heart failure they are usually transient.[64] Newman and Jacobson[48] described a case in which congestive changes occurred in the upper lobes, giving an appearance simulating pulmonary infiltrations of active pulmonary tuberculosis. These regions cleared completely in a few days after treatment for cardiac failure. In explanation of the unusual location of the congestive changes, they suggested that it may have been due to the fact that the patient had evidence of considerable emphysema in the bases of both lungs.

Vascular Congestion in Left-to-Right Shunts.[45] Conditions of main practical interest at this time are the septal defects and patent ductus arteriosus. In these conditions, when the left-to-right shunt predominates and the flow of blood through the lungs is large, vascular congestion becomes obviously manifested. The terms "increased blood flow" or "hyperemia" are probably preferable to "congestion," since congestion usually implies some degree of cardiac failure. The appearance of the vascular tree in conditions of increased blood flow is quite different from its appearance in congestion owing to cardiac failure in that, in the former condition, the pulmonary vasculature is distinct—vessels are plumper than normal and they are easily identified. It is only when cardiac failure is superimposed that the characteristic haziness is added to the roentgenologic picture; but even then the picture usually remains

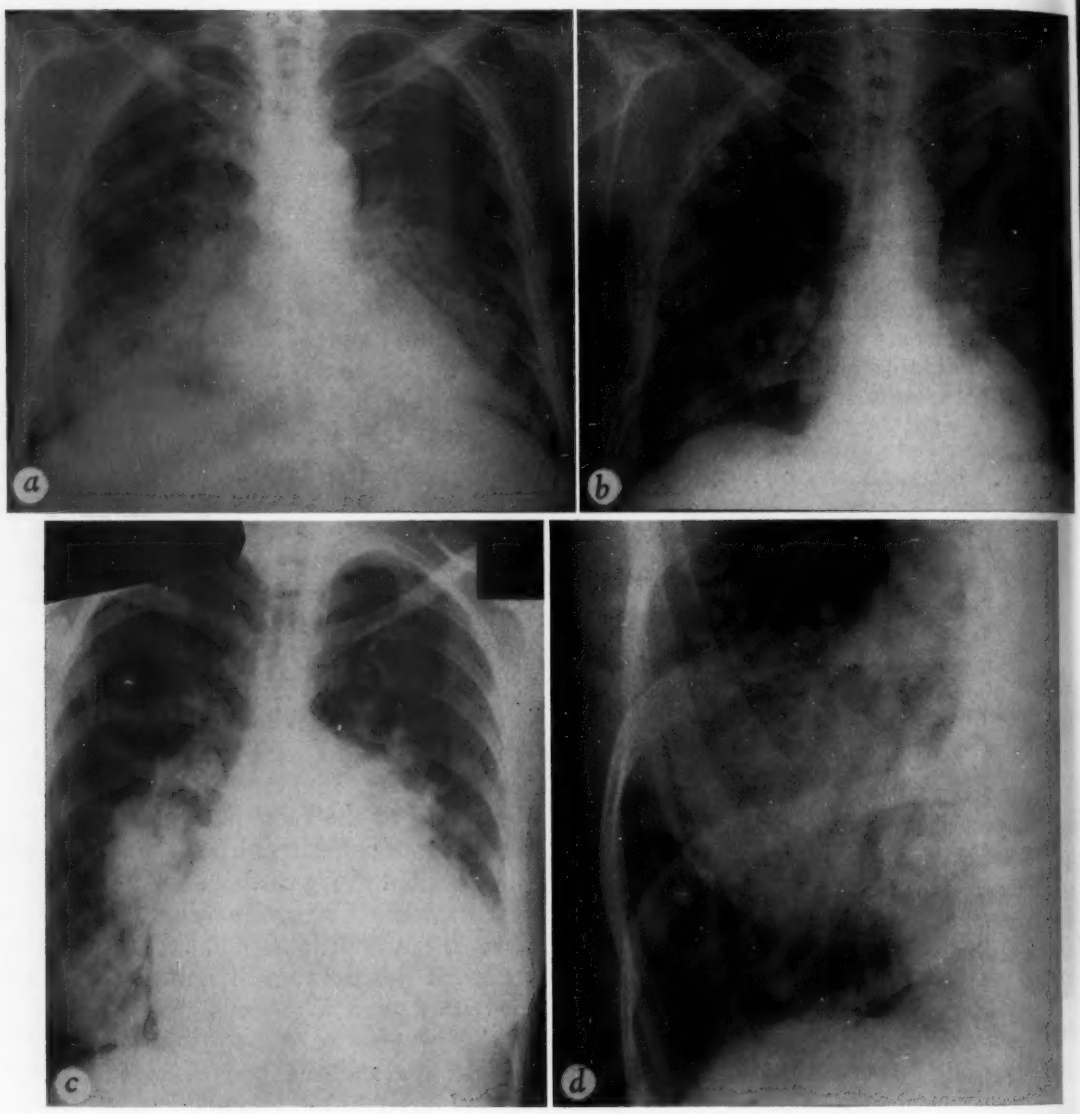


FIG. 1. (*a*) Posteroanterior view of thorax of a patient with coronary sclerosis, cardiac decompensation and advanced basilar pulmonary congestion. (*b*) Two days later, after rest in bed and digitalization, the pulmonary congestion showed considerable evidence of clearing. (*c*) Posteroanterior view of thorax of a fifty-five year old woman with an atrial septal defect and severe pulmonary hypertension. Note huge hilar arteries and disproportionately small peripheral pulmonary vessels. (*d*) Detail of lower field of right lung of patient with ventricular septal defect, showing decided accentuation of vascular markings.

distinct from that in cases of acquired or degenerative cardiac disease previously discussed under vascular congestion in cardiac failure (Fig. 1, *c* and *d*).

Occasionally a left-to-right shunt may be so overwhelming, particularly in some cases of ventricular septal defect, that the patient practieally "drowns" in his own blood. In such patients patches of pulmonary consolidation are frequently seen in the roentgenograms (Fig. 2, *a* and *b*). Although these pulmonary lesions are partly due to an inflammatory process, they are also partly the result of abnormal physiology, and this is attested to by the marked improvement the lungs undergo after a simple (but now outmoded) banding procedure on the pulmonary trunk (Fig. 2, *c* and *d*).[13,19,47]

Of interest are the rare cases of myo-

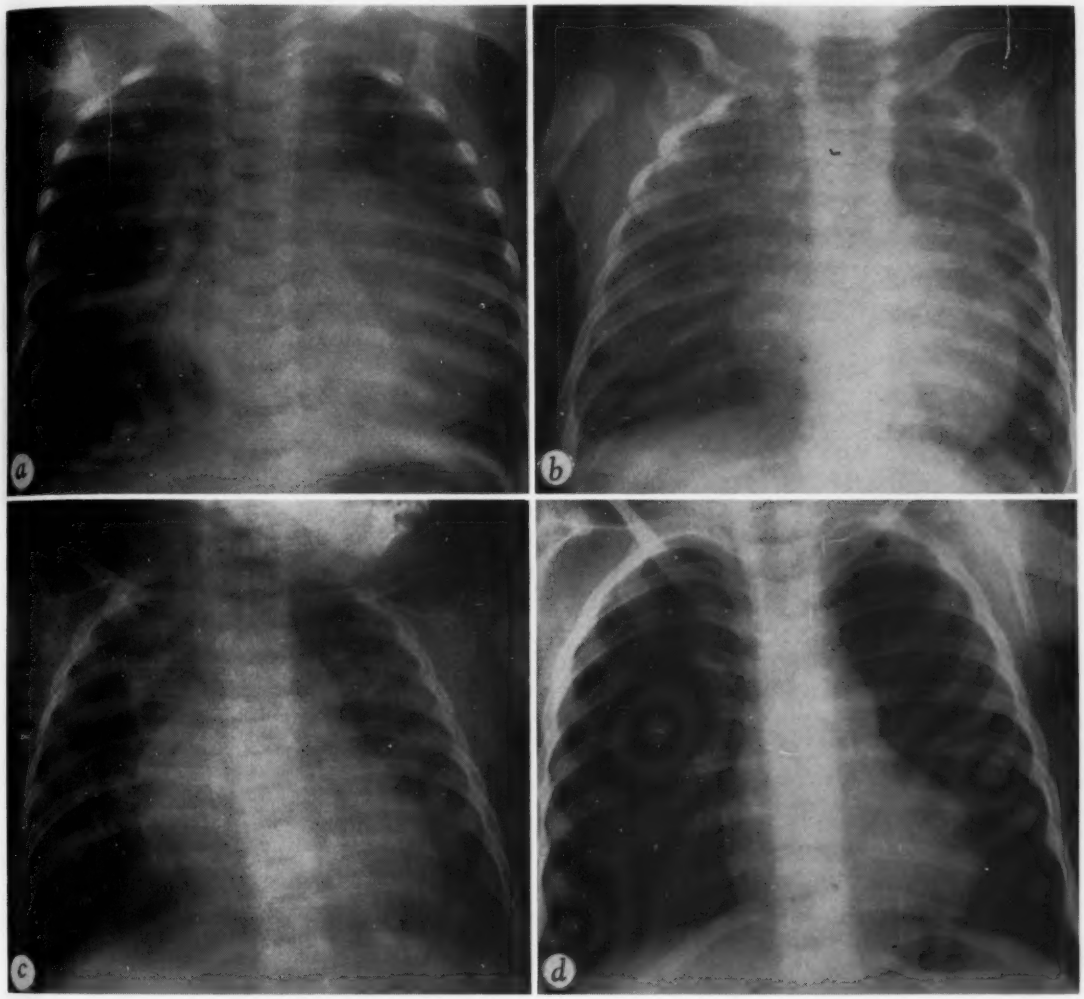


FIG. 2. (*a*) Posteroanterior view of thorax of child with a ventricular septal defect and corrected transposition. Note consolidation and collapse of the right middle and left lower lobes, and advanced cardiomegaly. (*b*) Five days after medical therapy both lobes showed evidence of re-expansion. (*c*) Posteroanterior view of thorax of patient with large ventricular septal defect and large shunt through the lung. Note evidence of pneumonitis in upper field of right lung. (*d*) Roentgenogram of same patient taken about three years after banding of the pulmonary artery which was done as a lifesaving procedure with a view to subsequent definitive correction.

cardial infarction with rupture of the interventricular septum in which the patient survives long enough to show roentgenologic manifestations of a left-to-right intracardiac shunt. We have examined such a patient fluoroscopically and noted considerable increase in pulmonary arterial activity similar to that which may be seen in congenital ventricular septal defect.

Lymphatic Congestion. We refer specifically to the costophrenic septal lines (Kerley's B lines)[10,39,63] that are so characteristic in pulmonary venous hypertension, usually in mitral heart disease, and occasionally in aortic valve disease with left ventricular failure and elevation of the pulmonary venous pressure. It is important to maintain strict criteria for these lines, since they are highly accurate diagnostic factors (Fig. 3).

The costophrenic regions probably are not the only regions in the lungs where lymphatics become congested in mitral heart disease. Congestion probably occurs

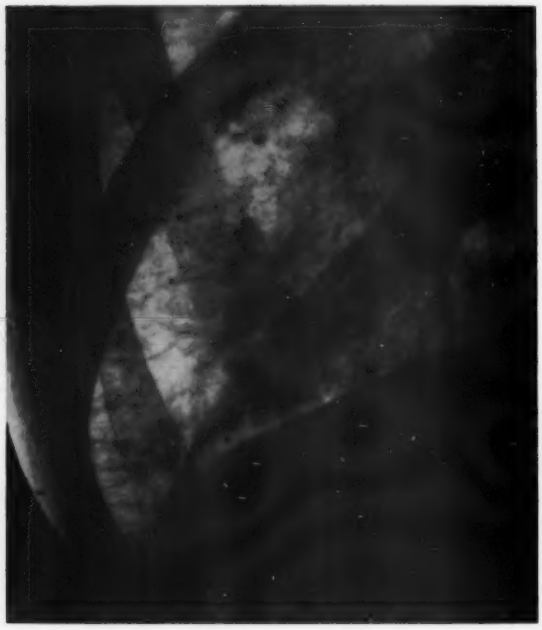


FIG. 3. Detail of posteroanterior roentgenogram of thorax of patient with severe mitral stenosis. Note the septal lines in the costophrenic angle. Compare with prominent vessels in Figure 1*d* from a patient with a ventricular septal defect.

throughout the lungs, but becomes more obvious in the costophrenic angles because of factors such as structural arrangement, lack of overlying shadows, and hydrostatic factors.

It is of interest, as Zdansky[78] pointed out, that hilar lymph nodes may enlarge as a result of lymphatic congestion in conditions such as mitral stenosis (Fig. 4, *a* and *b*).

Pulmonary Hemosiderosis. We refer here only to the hemosiderosis of mitral heart disease and not to the so-called idiopathic type of hemosiderosis which probably has its origin in a hypersensitivity state. In occasional cases of mitral stenosis roentgenograms show multiple nodular shadows throughout both lungs. The shadows usually average about 2 to 5 mm. in diameter, and are due to numerous deposits of hemosiderin throughout the lungs (Fig. 5). It is true that in most cases of significant mitral stenosis the tissues of the lungs show microscopic evidence of increased deposits of hemosiderin, but in the cases under discussion, a condition occurs which is evident roentgenographically.

This condition is rare, and is the result of intra-alveolar aggregations of hemosiderin-containing macrophages and secondary fibrotic reaction. The resulting roentgenographic appearance may simulate miliary tuberculosis or pneumoconiosis.[27,52] These opacities do not alter in appearance over long periods.[62]

In arriving at the diagnosis, other mani-

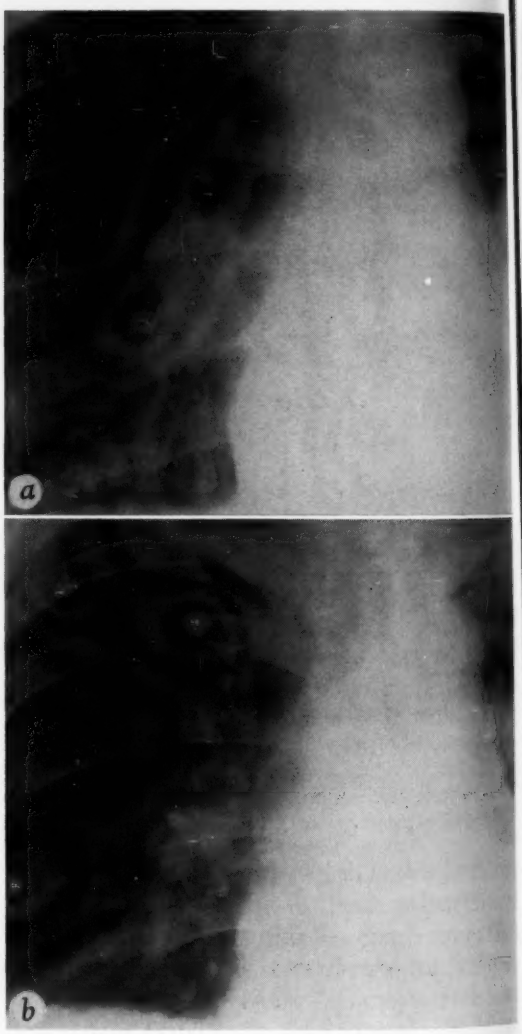


FIG. 4. (*a*) Posteroanterior view of thorax of patient having severe mitral stenosis. Note the size of the right hilar shadow, the appearance of which could easily be mistaken for enlarged lymph nodes. (*b*) This roentgenogram was made three weeks after commissurotomy was performed.

Fig. 5. Section of posteroanterior view of thorax of patient with severe mitral stenosis. Note nodular shadows in the lung field due to hemosiderosis.

festations of mitral heart disease, such as the cardiac configuration and clinical signs of mitral disease, will make the diagnosis evident.

Pulmonary Ossification in Mitral Stenosis. Even rarer than roentgenographic evidence of pulmonary hemosiderosis in mitral heart disease is the occurrence of disseminated pulmonary ossification in mitral disease associated with chronic passive congestion.[42] Although we have encountered 4 cases of mitral stenosis with evidence of disseminated calcific lesions in the lungs, we do not have histologic proof of formation of bone in these lesions.

Pulmonary Embolism and Infarction. This may be a manifestation of several types of cardiac disease, usually myocardial infarction and bacterial endocarditis. Practically every case differs in roentgenographic appearance.

According to Smith,[65] "The diagnosis of an infarct by x-ray examination is probably subject to more error than any other lesion of the lungs; the error is almost always a negative one, i.e., failure to consider an infarct as the cause of the abnormal shadow."

Zdansky[78] pointed out that pulmonary infarcts are unequivocally recognized only in the minority of cases. Usually there is so much associated pulmonary congestion that the shadow of the infarct cannot be outlined (Fig. 6*a*). The appearance also depends on the plane of projection. It should be remembered that pulmonary infarction may be accompanied by pleural effusion and even empyema. In general the lesion will appear similar to a rather amorphous area of consolidation affecting a portion of the lung. Kirklin and Faust[40] found a definite predilection for occurrence of the lesion in the right lower lobe. The textbook infarct which has a triangular shape (Fig. 6*b*), the triangle representing the involved segment of pulmonary tissue with its base on the pleural periphery and its apex toward the hilus, is indeed a roentgenographic rarity.

Keating and co-workers[38] have described several interesting cases of "chronic massive thrombosis of the pulmonary arteries." Roentgenologically their cases were extremely interesting, the changes in the central pulmonary arteries closely simulating enlarged hilar lymph nodes.

After resolution of a pulmonary infarct there may be a complete return to normal, or merely streaky or linear fibrotic shadows may be evident on the roentgenogram.

Acute Pulmonary Edema. Many times pulmonary edema, no doubt, occurs in conjunction with pulmonary vascular congestion, without giving any characteristic roentgenographic appearance. However, occasionally it occurs in the acute form; it is easily recognized on the roentgenograms and is described by names such as "bats-wing," "angel wing," "butterfly wing," or

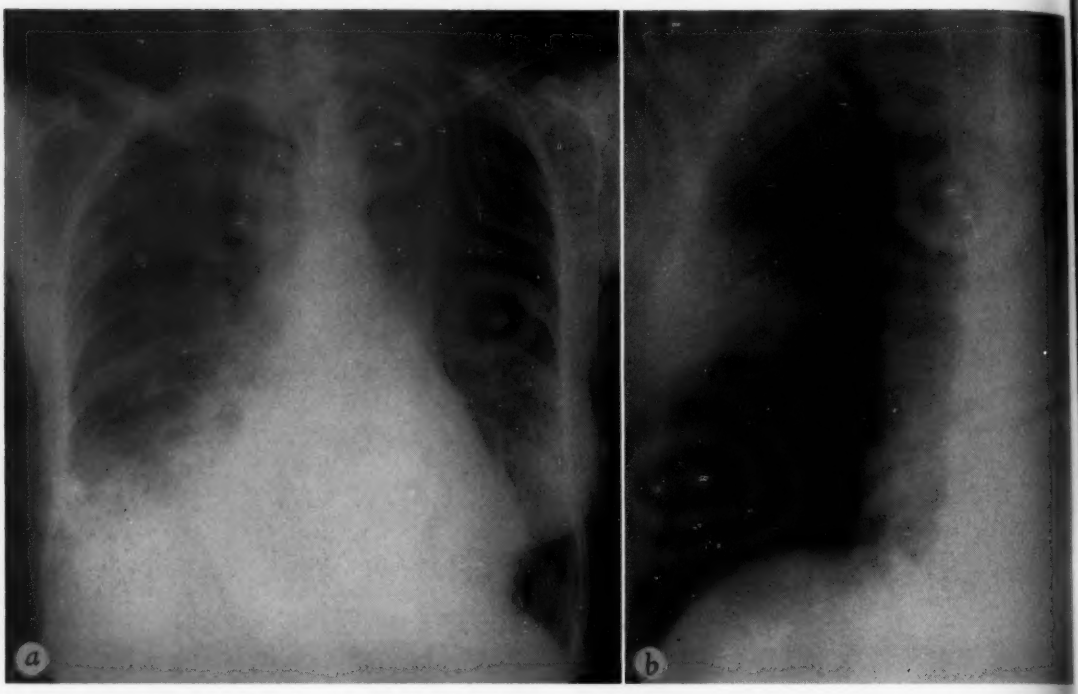


FIG. 6. (*a*) Posteroanterior view of thorax of a fifty-two year old woman with mitral heart disease who died after development of numerous emboli including one in the base of the right lung. This is the more usual appearance of pulmonary embolism and infarction. (*b*) Posteroanterior view of a pulmonary infarct in a patient who had a long history of coronary infarcts. The triangular textbook appearance is the exception rather than the rule.

"uremic pneumonia" which usually describe its appearance (Fig. 7*a*). Characteristically the inner and middle thirds of the lung (medulla) undergo a dense, frosted-glass opacification, while the outer thirds (mantle) of the lung fields remain clear.

This condition almost always accompanies an acute attack of dyspnea which complicates an illness in which strain is being thrown on the left side of the heart. Hodson[33] stated, "The blood urea need not be raised above normal limits at the time of the lesion's formation." Rendich, Levy and Cove[56] did not find an example of "batswing" pulmonary edema in 50 cases of severe uremia selected at random.

A failing left heart appears to bear a much more important causal relationship to batswing pulmonary edema than does uremia.[33,36] Gravity apparently does not affect the lesion. The mechanics of this condition are uncertain. Rarely, and for obscure but probably interesting reasons, acute pulmonary edema may manifest itself on the roentgenogram as a unilateral process (Fig. 7, *b* and *c*). However, the characteristic roentgenologic texture in such cases is identical to the commoner bilateral condition.

Decreased Pulmonary Vascularity. Only when this is marked and evident, so that no hesitation need occur in labeling the lungs as "anemic," is this condition of practical interest from the roentgenologic vewpoint. Such cases usually fall into one of the following categories: tetralogy of Fallot, severe pulmonic stenosis, Ebstein's malformation of the tricuspid valve, or tricuspid atresia. The exact diagnosis is usually clinical, but can be assisted by angiocardiography or cardiac catheterization, or both. Figure 8 is an example of the anemic type of lung fields that may be encountered in certain cases of tricuspid atresia.

Enlarged Bronchial Arteries. Enlargement of the bronchial arteries often occurs in pa-

Fig. 7. (*a*) So-called batswing or angel-wing densities in the lungs due to acute pulmonary edema in a patient with malignant hypertension, cardiac and renal failure and moderate elevation of blood urea. (*b*) Posteroanterior view of thorax of a thirty-four year old woman with a thirty year history of diabetes and evidence of coronary insufficiency. Note evidence of acute unilateral pulmonary edema. (*c*) Roentgenogram made three days after Figure 7*b*. Death occurred eleven months later of coronary thrombosis.

Fig. 8. Posteroanterior view of thorax of patient with tricuspid atresia. Note marked clarity of the lung fields due to poor vascularization.

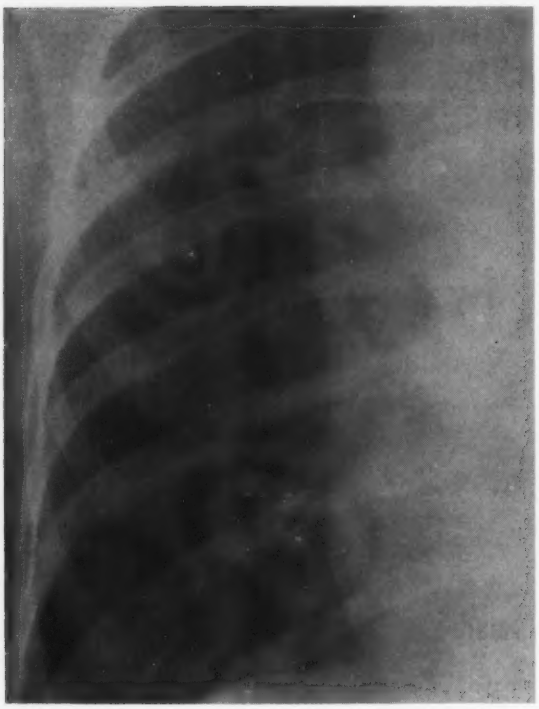


FIG. 9. Portion of posteroanterior roentgenogram showing irregularly branching bronchial arteries in a case of tetralogy of Fallot.

tients in whom the normal path of pulmonary vascularization, that is, the pulmonary arteries, is obstructed. Roentgenologically the enlarged bronchial arteries present irregular branching pathways, quite unlike the normal (Fig. 9) orderly branching of pulmonary arteries.[12] End-on views may present multiple punctate shadows.

Anomalous Venous Connection of the Right Lung. This condition should be suspected when an anomalous vessel is seen in the right lower lung fields, and when it appears to take a course toward the inferior vena cava. Laminagraphy and angiocardiography are good methods of corroborating the diagnosis[7,18] (Fig. 10*a*). Of special interest (Fig. 10, *b*, *c* and *d*) is the occasional occurrence of partial agenesis of the right lung in these cases with the associated manifestations of a shift of the heart toward the right hemithorax and a smaller appearance of the right hemithorax compared to the left.[6,28]

Intralobar Bronchopulmonary Sequestration.[54] Characteristically this condition presents as a solid or cystic lesion in the posterior basal segment of the lung. It receives an arterial supply from an anomalous branch of the descending thoracic or high abdominal aorta, a vessel which, according to some theories, bears an etiologic relationship to the condition (Fig. 11, *a* and *b*).

The condition is usually discovered as a result of a routine thoracic roentgenogram in an asymptomatic patient, or following investigation of recurrent bouts of pulmonary inflammatory disease.

Pulmonary Arteriovenous Fistula. This familial disease is frequently part of a more widespread developmental anomaly of the vascular system known as hereditary hemorrhagic telangiectasia, Babington's disease,[30] or Rendu-Osler-Weber disease. It may be manifested in the roentgenogram as a round or a lobulated mass in any part of the lung. Usually the vessels supplying and draining the lesion can be seen to connect with the mass (Fig. 12*a*). Excellent roentgenologic corroboration of the lesion can be obtained by laminagrams (Fig. 12*b*) or angiopneumograms or both.[71] Not infrequently there will be more than one arteriovenous fistula in the lungs. According to Tobin and Wilder,[74] "If any surgical procedure is planned, the proper assessment of the number and size of various fistulas is of the utmost importance." Other clinical corroboration such as hereditary hemorrhagic telangiectasia of the mucosa of the oral cavity, the nasal cavity, or the lips may be evident.

Symptoms and signs may include cyanosis, clubbing of the fingers and toes, polycythemia, hemoptysis, evidence of hemorrhagic telangiectasis and continuous murmur over a pulmonary tumor. Fluoroscopic examination may reveal pulsations in the mass, and a difference in size is frequently observed between the Valsalva and Müller maneuvers.

PLEURAL ENTITIES

Common Basilar Pleural Effusions. These effusions are common in heart disease with failure. For some reason they are more com-

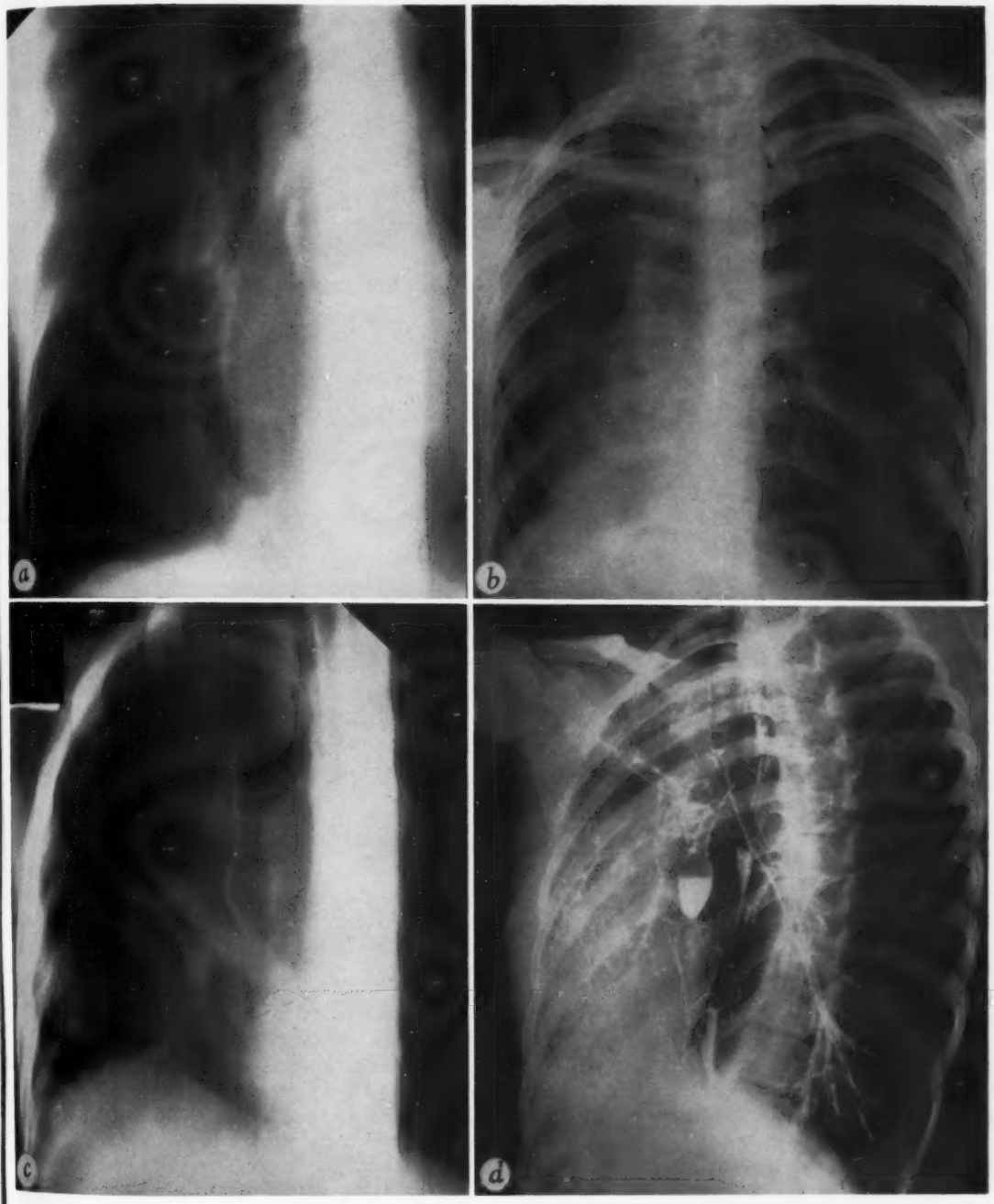


Fig. 10. (*a*) Laminagram of lower field of right lung showing an anomalous vein draining the right lung to the inferior vena cava. (*b*) Posteroanterior view of thorax of a woman showing dextrodisplacement of the heart and a small right hemithorax. (*c*) Laminagram shows anomalous vein draining right lung and cystic hypogenesis of a bronchus, probably the lower lobe bronchus. (*d*) Bronchogram in right posterior oblique projection confirms the cystic hypogenesis of the right lower lobe bronchus.

mon on the right side. Their roentgenologic appearance is familar to all physicians. Perhaps the only comment that need be made here is that occasionally the necessity arises to differentiate these effusions from pleural thickening, and lateral decubital views will be of value in the differentiation.

It is of interest to mention here the case described by Newman and Jacobson.[48] A patient having cardiac failure had been ly-

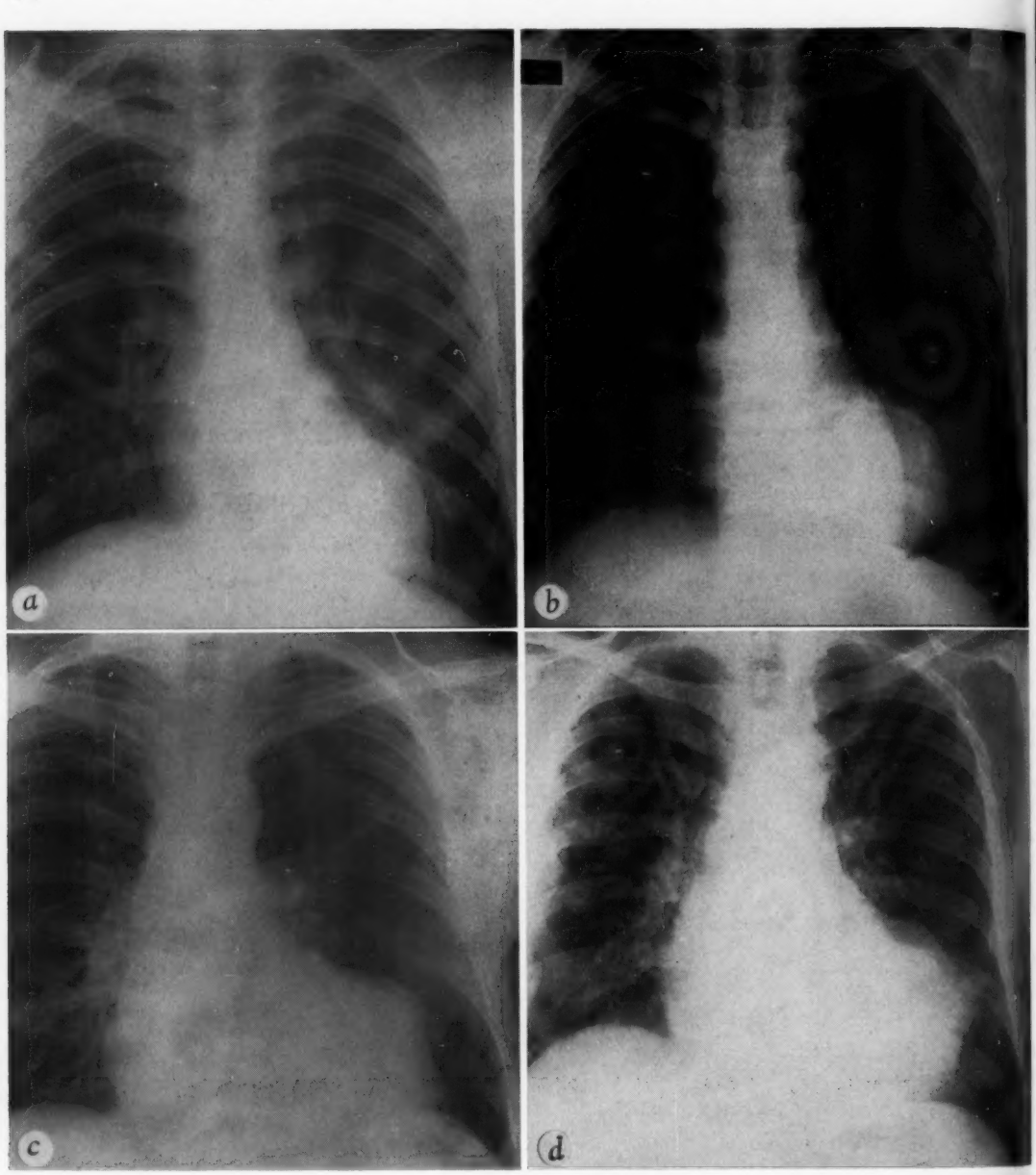


Fig. 11. (*a* and *b*) Routine and Bucky posteroanterior roentgenograms of thorax of a patient with sequestration cyst of the posterior basal segment of the left lung. Note that the shadow of the heart is well seen through the cyst shadow, and compare with the case of ventricular aneurysm. (*c* and *d*) Posteroanterior view of thoraces of 2 patients with cardiac aneurysms following myocardial infarction. Note the typical irregular contour of the aneurysm at the lower left heart border in each case.

ing on his right side for a considerable time, with the result that much edema had occurred on this side including the right hemithorax. The thoracic roentgenogram showed a complete opacification of the right hemithorax, as if by a massive pleural effusion. Two days after the patient had been encouraged to lie on the left side, the lung fields appeared clear as a result of a more general distribution of the anasarca.

Uncommon Pleural Effusions. Loculated Interlobar Effusions. These effusions are of interest in that they may, owing to their sharply defined loculation in the oblique or

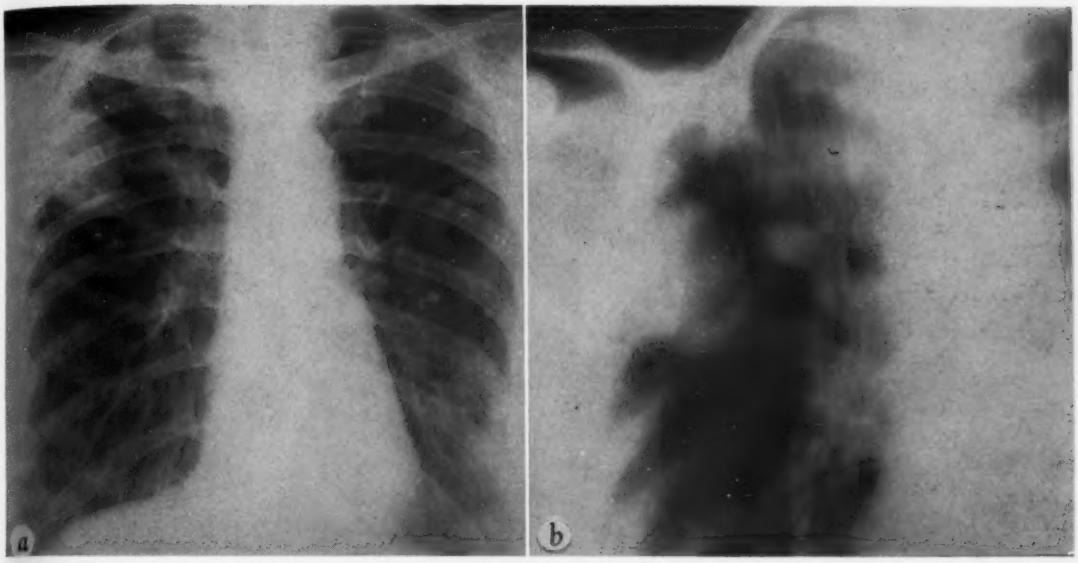


FIG. 12. (*a*) Posteroanterior view showing pulmonary arteriovenous fistula in right upper lobe. (*b*) Laminagram clearly shows connecting vessels.

horizontal fissures, appear similar to a mass on the posteroanterior roentgenograms.[32,48,72] A lateral view will usually identify the interlobar location of the mass. These effusions occur during bouts of cardiac decompensation, and will disappear rapidly and dramatically during rest in bed and digitalization, thus accounting for the designation "vanishing tumor" (Fig. 13, *a* and *b*). It is interesting that when decompensation recurs, these effusions tend to recur in the same location.

Excellent diagrammatic representations of the various types of pleural effusion (costal, diaphragmatic, mediastinal, interlobar) are provided in the sixth edition of the textbook of Schinz *et al.*[59]

Mediastinal Pleural Effusion.[61] The location of pleural effusion in relation to some part of the mediastinum, but sparing apparently the basal and peripheral regions, can give rise to unusual appearances on the posteroanterior roentgenogram. If located below the level of the pulmonary ligament, such an effusion may, as Rigler[57] has pointed out, simulate the appearance of a collapsed lobe. In the example we present (Fig. 13, *c* and *d*) the effusion occurred in the posterior mediastinal pleural space and along the paravertebral gutter, giving rise to a very unusual picture. With rest in bed and digitalization, the effusion rapidly disappeared.

Subpulmonary Pleural Effusion. These effusions remain between the base of the lung and the diaphragm, a sharp costophrenic angle being maintained. The appearance therefore simulates an elevated hemidiaphragm, and decubital views are required to reveal the true nature of the lesion.

ENTITIES OF BONY THORAX

Coarctation (Fig. 14). One of the best-known roentgenologic signs of cardiovascular disease is the notching of the ribs seen in cases of coarctation (Fig. 14*b*). Roentgenograms of approximately 80 per cent of patients with coarctation will show evidence of notching of ribs. Notching, as is well known, is the result of increased size and tortuosity of the intercostal arteries resulting in localized erosion of the costal grooves. This sign of coarctation was first described in the roentgenologic literature by Railsback and Dock in 1929.[55]

Intercostal Arteriovenous Malformation. Occasionally, an arterial malformation will be situated so that it implicates the peripheral thoracic vessels, and in such a case

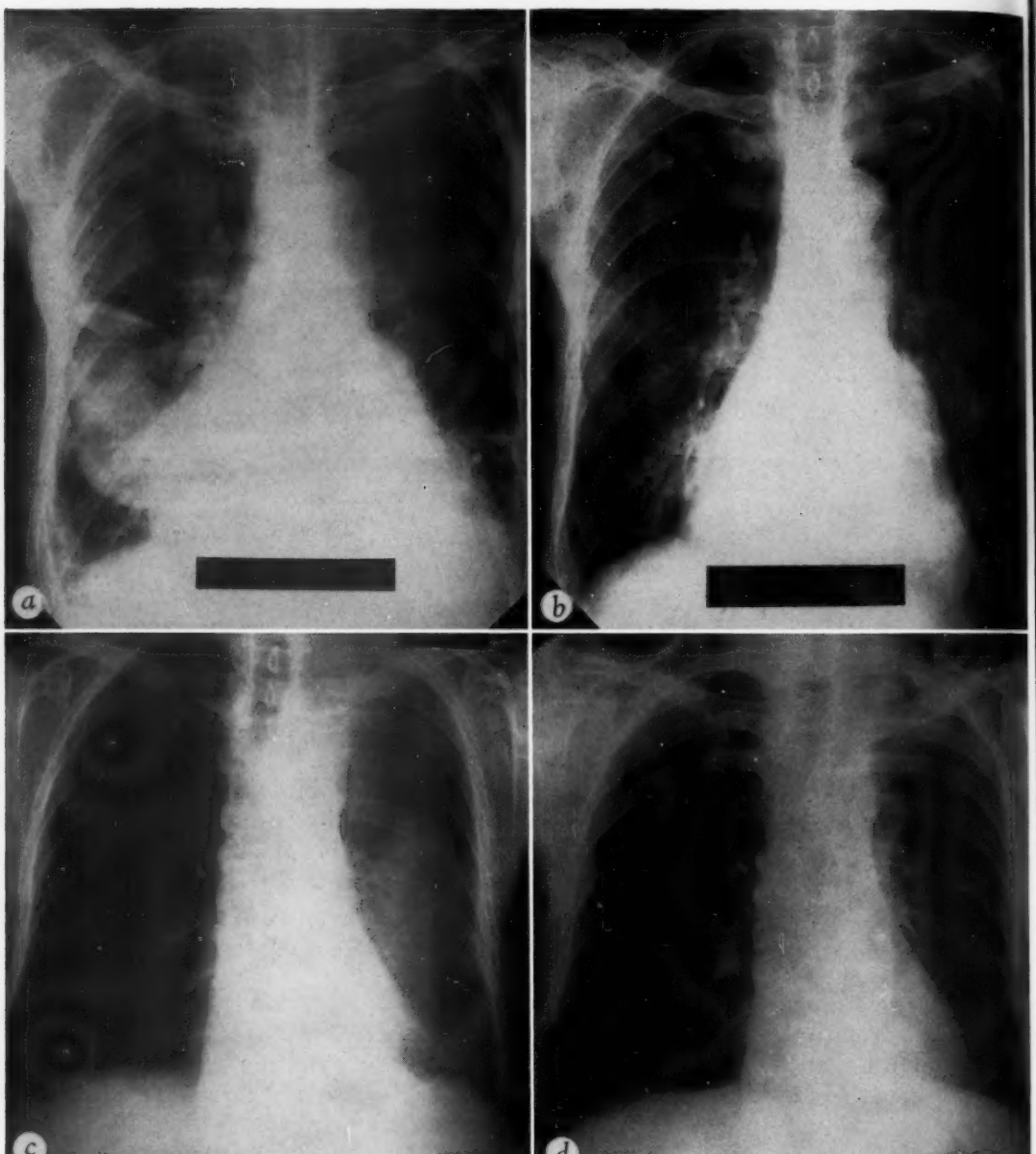


FIG. 13. (*a*) Posteroanterior view showing cardiac enlargement and interlobar effusions; (*b*) four days after rest in bed and digitalization for mitral heart disease. "Vanishing tumor." (*c*) Posteroanterior view showing mediastinal pleural effusion; (*d*) eleven days after rest in bed and digitalization for cardiac decompensation.

localized rib notching may occur. In a patient with localized rib notching and a pulmonary lesion (Fig. 15), an attempt should be made to hear a murmur in the region of the rib notching. Probably the best way in which to demonstrate the nature of such a lesion is by means of angiocardiography. Maier and Stout[44] described a patient with a precordial arteriovenous fistula in whom the roentgenogram showed questionable evidence of phleboliths in the region of the lesion.

Aneurysms. An aneurysm of the innominate artery may resemble the shadow of an

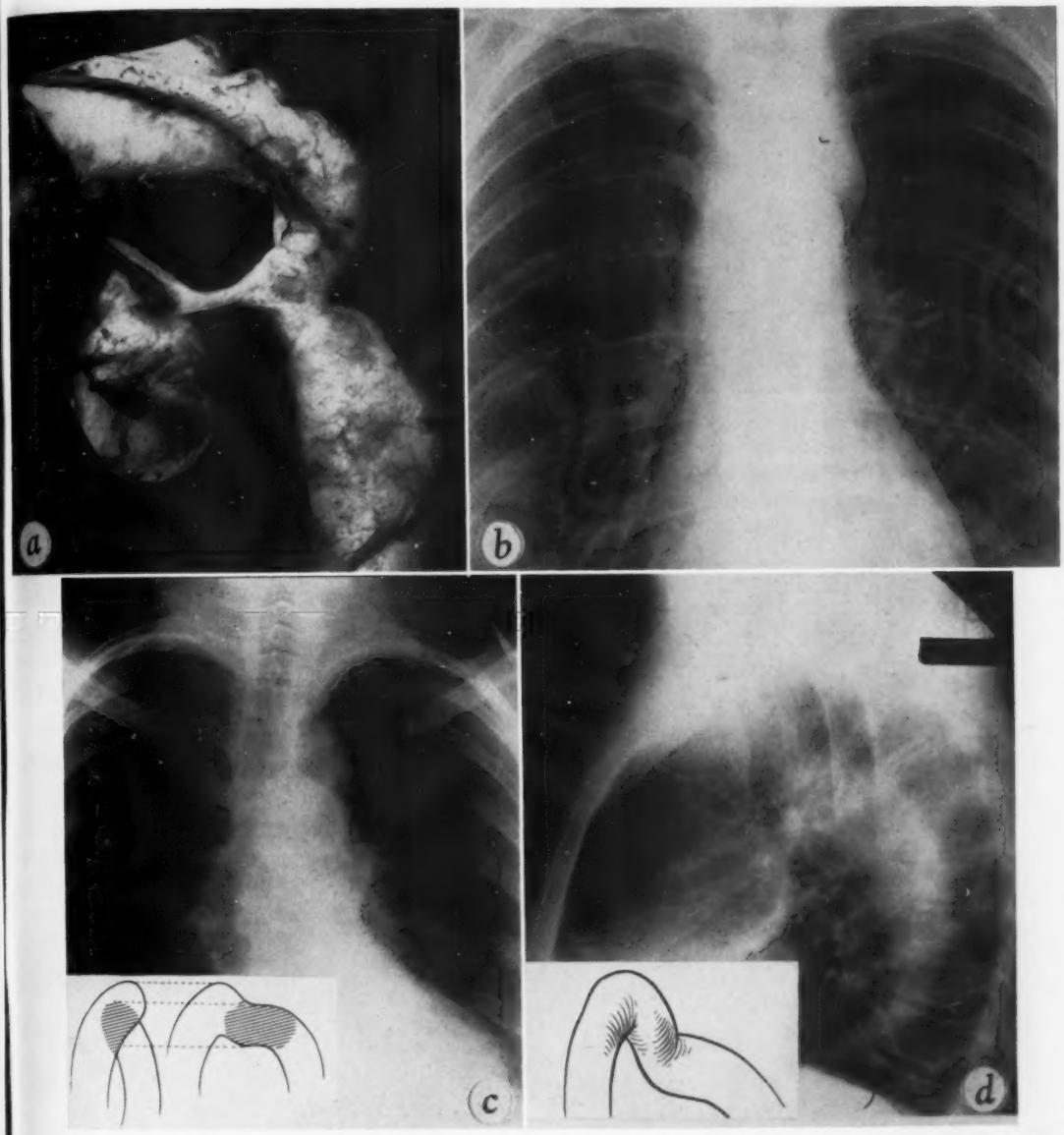


FIG. 14. (*a*) Gross specimen of coarcted segment of aorta. (*b*) Posteroanterior view of thorax showing notched aorta in coarctation; note rib notching. (*c* and *d*) Posteroanterior and lateral views illustrating "double aortic knob" due to kinking of aortic arch, accentuated by tortuosity of descending thoracic aorta.

enlarged thyroid gland on a posteroanterior roentgenogram, in that it causes lateral displacement of the trachea. But an aneurysm of the innominate artery is almost invariably accompanied by aneurysmal formation at the arch of the aorta. Furthermore, innominate aneurysm may cause characteristic erosion of the medial end of the clavicle or of the manubrium (Fig. 16).

Aneurysms of the arch and descending thoracic aorta also may cause painful erosion of the sternum or of the anterior surfaces of dorsal vertebrae.

MEDIASTINAL MANIFESTATIONS OF CARDIOVASCULAR DISEASES

Conditions Affecting the Aorta and Its Brachiocephalic Branches. Coarctation of

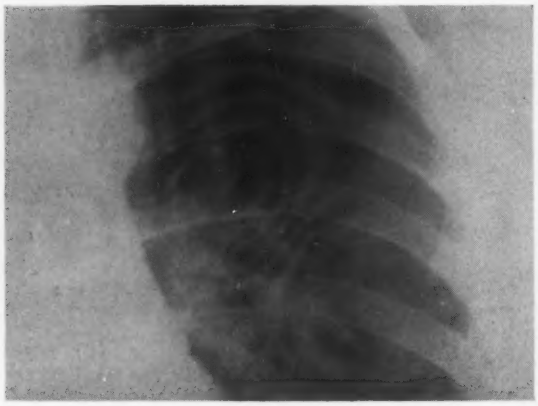


Fig. 15. Posteroanterior view showing aneurysm of aortic arch and of innominate artery. Note lateral displacement of trachea and erosion of superior aspect of medial end of clavicle.

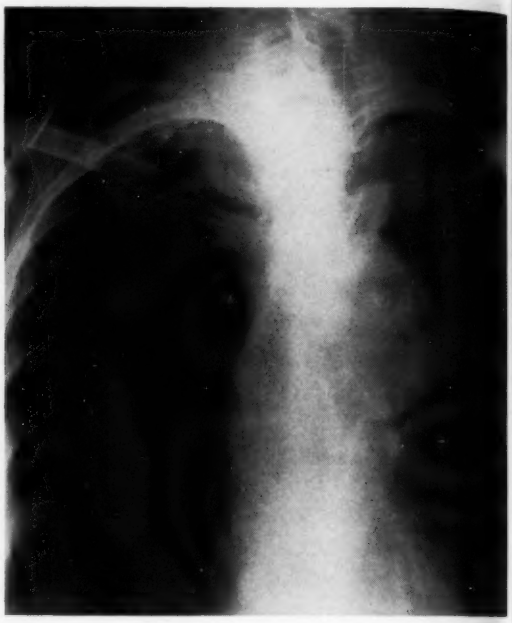


Fig. 16. Irregular lesion in upper part of left lung associated with notching of the sixth rib. This was an arteriovenous malformation involving also the intercostal vessels at this level.

Aorta. In approximately a third of patients with coarctation of the aorta, the actual site of coarctation can be visualized.[11] It may be very obvious or it may require careful study of the roentgenogram (Fig. 14, *a* and *b*). The notch is best seen in the posteroanterior projection and represents the actual site of coarctation. The segment above it may be composed of the dilated left subclavian artery or the descending portion of the aortic arch—there is no way of establishing this except by aortography or angiocardiography, but from the point of view of diagnosis this is unimportant.

The notch deformity of the aorta may be so pronounced that the component above the notch simulates a mediastinal mass.

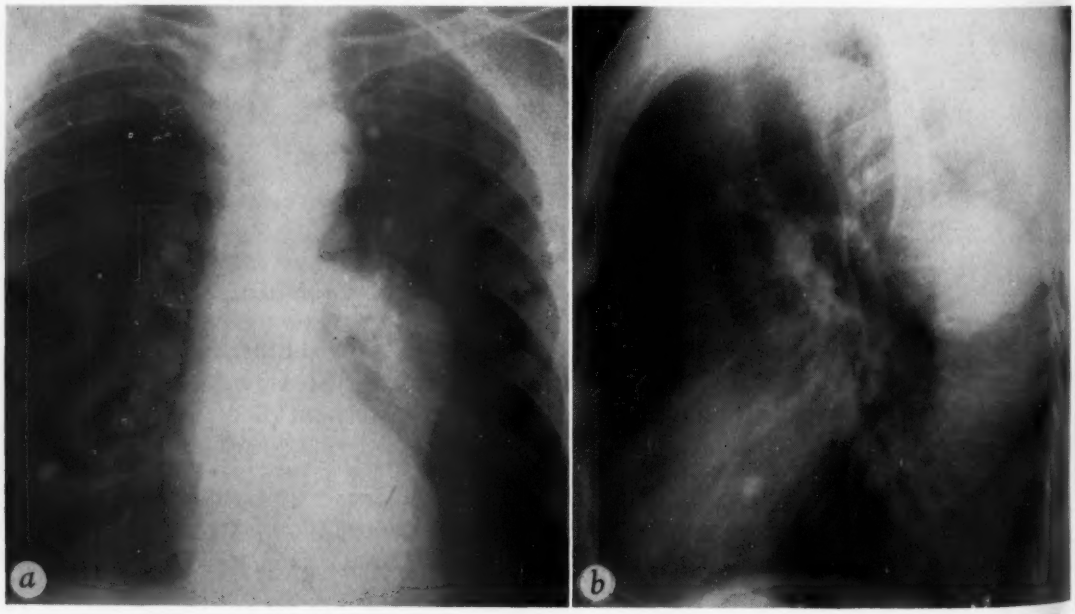


Fig. 17. (*a* and *b*) Posteroanterior and lateral views showing buckled and dilated thoracic aorta resembling a mass in the posteroanterior view.

Familiarity with such an appearance in coarctation of the aorta, together with pertinent clinical and other roentgenologic findings will lead to the correct diagnosis.

Kinking of Aortic Arch.[8,17,68] On a roentgenogram this benign developmental anomaly of the aortic arch may easily be mistaken for a mediastinal mass or coarctation of the aorta (Fig. 14, *c* and *d*). The appearance is the result of a buckle in the aortic arch in the region of insertion of the ligamentum arteriosum. The term "pseudocoarctation" has been appended to this condition, because the roentgenologic picture may resemble marked aortic notching in coarctation. However, there is no clinical evidence of coarctation in these cases, and other roentgenologic manifestations of coarctation are absent. Because many of these patients are initially suspected of having a mediastinal tumor, the term "pseudotumor" may be applied logically but also erroneously in these cases. It should be noted that buckling of a tortuous aorta may likewise imitate a mediastinal mass (Fig. 17, *a* and *b*).

Right-sided Aortic Arch. A right-sided position of the aortic arch is usually detected with ease when a roentgenogram of good quality is obtained (Fig. 18*a*). The indentation of the tracheal shadow on the right rather than on the left is good proof of the location of the aortic arch, even in small children. Assmann[3] is credited by Paul[51] with having been the first to indicate that the right aortic arch with a right descending thoracic aorta can be diagnosed roentgenologically during life. Usually right-sided aorta causes no difficulty in roentgenologic evaluation.

Occasionally, however, variations of the roentgenologic appearance occur which cause diagnostic difficulty and may even result in serious inconvenience to the patient; for example, unnecessary thoracic exploration for suspected mediastinal mass. It is of paramount importance that any mediastinal "mass" that is not causing the patient any symptoms be thoroughly explored from the diagnostic viewpoint. Bucky roentgenograms, oblique views, a lateral view and fluoroscopy with a barium swallow may all be necessary in order to uncover an anomalous thoracic aorta masquerading as a mediastinal mass. Figure 18*b* is an interesting example of a right-sided aorta which passes to the left behind the trachea and the esophagus and then descends along the right border of the heart, simulating a large mass in the mediastinum. The Bucky roentgenogram indicates the deviation of the trachea to the left, and the barium swallow demonstrates the arch passing behind the esophagus (Fig. 18, *c* and *d*). A similar case was described by Szabados,[73] in which the correct diagnosis was arrived at by means of a barium swallow and oblique roentgenograms.

Figure 19, *a* and *b* illustrates a similar but slightly more deceptive case. Eibach[21] and Franke[24] illustrated similar cases in papers on the problem of thoracic aortic anomalies (Fig. 19*c*). Arkin[1] is said to have been the first to have described this type of anomaly.

We have referred to right-sided thoracic aorta only insofar as it may create a diagnostic problem with reference to the possibility of its being mistaken for a mediastinal mass. Outside the scope of this discussion are those cases in which the problem of accurate location of the aorta may be of assistance to the cardiovascular surgeon; for example, patients with tetralogy of Fallot or transposition of the great vessels.

Dextroposition of the Descending Thoracic Aorta. Snider, Gildenhorn and Rubenstein[67] described an interesting case in which an exploratory thoracotomy had been performed for a possible mass presenting in the right cardiophrenic angle on the posteroanterior reontgenographic view. It turned out that the so-called mass was the greatly elongated descending thoracic aorta which had crossed anteriorly to the spinal column into the right hemithorax before returning to the left hemithorax. They described 2 other such cases in their communication, in both of which the identity of the shadow was established by additional roentgenologic procedures.

An interesting example of this entity was

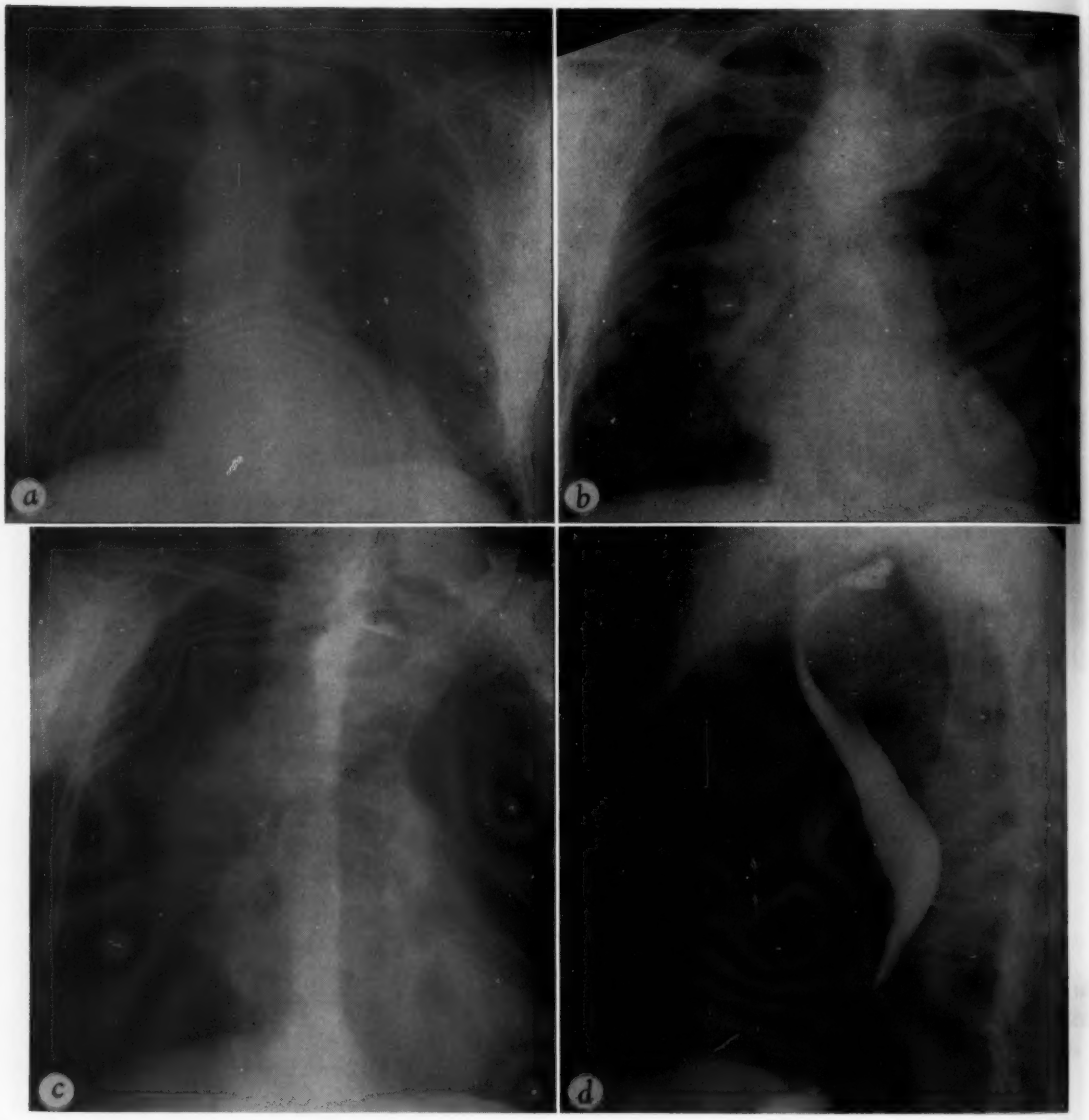
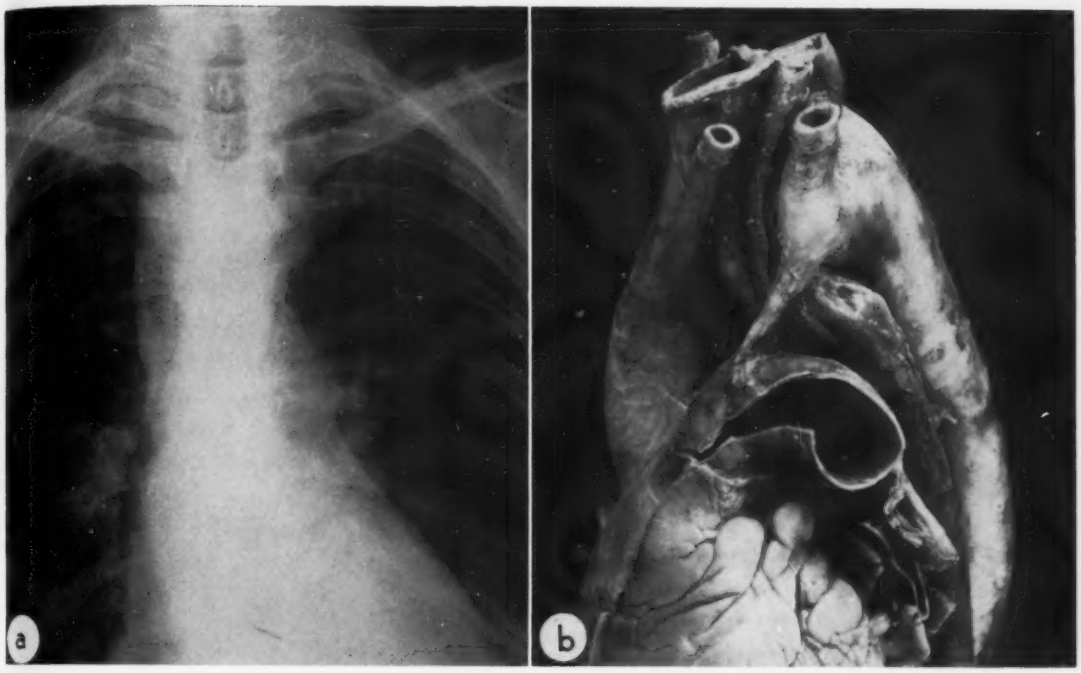


FIG. 18. (*a*) Posteroanterior view of thorax showing right aortic arch. Note tracheal shadow indented on the right. (*b*) Posteroanterior view of patient with right-sided aortic arch which passes behind esophagus and descends on the right. (*c*) Bucky roentgenogram of same case clearly shows descending aorta on right. (*d*) Barium swallow shows aortic knob behind esophagus.

recently brought to our attention by Dr. Milton W. Anderson of the Section of Cardiology of the Mayo Clinic. A posteroanterior view of the thorax of a patient with Cushing's disease and recent severe hypertension gave the appearance seen in Figure 20*a*. Besides the cardiac enlargement, the Bucky roentgenograms indicated that the thoracic portion of the aorta descended to the right of the spinal column, giving the appearance of a mass or of cardiac enlargement to the right. Of interest was the marked shortening of the aorta following adrenalectomy and the return of the blood pressure to normal (Fig. 20*b*). Figure 20*c* is a diagram of a similar case from the article by Franke.[24]

The findings in this case would seem to contradict the suggestion by Snider and co-workers[67] that systolic hypertension in



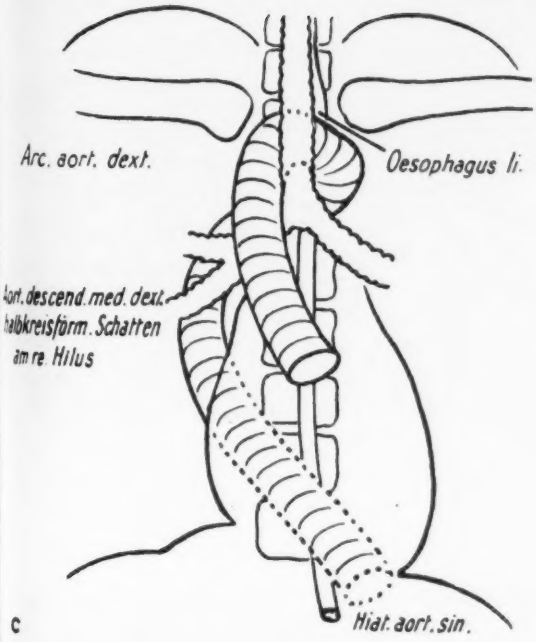


FIG. 19. (*a*) Posteroanterior view of a patient with a right aortic arch with retro-esophageal "diverticulum," and right descending aorta. (*b*) Gross specimen of same case, seen from the left. (*c*) Diagram of a somewhat similar situation (from Franke, Hans: Über Entwicklungs- und Lageanomalien der Aorta. *Fortschr. a. d. Geb. d. Röntgenstrahlen ver. m. Röntgenpraxis.* Georg Thieme Verlag, Stuttgart, 1950, *73*, 267–280. Reproduced by permission.).

one of their patients was the result rather than the cause of the unusual aortic course. It also throws light on a problem raised by Corcoran and Coleman.[14] In one of their cases the descending thoracic aorta showed a marked retrocardiac buckle, simulating a tumor. The patient's blood pressure was 172 mm. of mercury systolic and 110 mm. diastolic. They favored, as an explanation of the aortic appearance, a developmental elongation because "it seems unlikely that elongation in adult life sufficient to produce the extreme tortuosity found in this case could occur in the relatively short segment

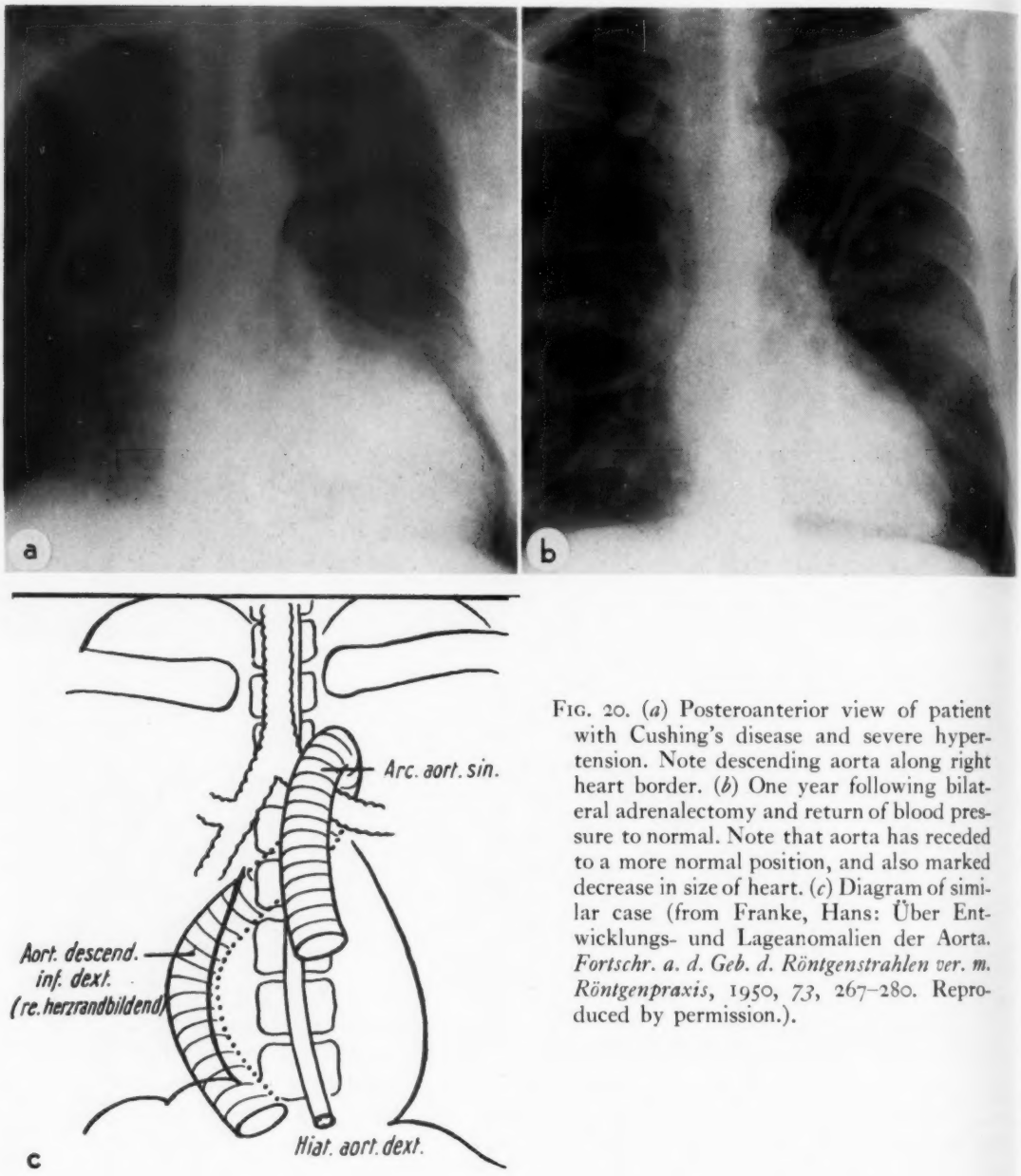


FIG. 20. (*a*) Posteroanterior view of patient with Cushing's disease and severe hypertension. Note descending aorta along right heart border. (*b*) One year following bilateral adrenalectomy and return of blood pressure to normal. Note that aorta has receded to a more normal position, and also marked decrease in size of heart. (*c*) Diagram of similar case (from Franke, Hans: Über Entwicklungs- und Lageanomalien der Aorta. *Fortschr. a. d. Geb. d. Röntgenstrahlen ver. m. Röntgenpraxis*, 1950, *73*, 267–280. Reproduced by permission.).

of the aorta lying between the heart and the diaphragm."

The anomaly of left aortic arch with right descending aorta was originally described by Paul.[51] Both of his patients were cyanotic cardiacs. Heinrich and Tamayo[31] have reported the condition in an otherwise asymptomatic adult, and Desai[15] has observed a striking variation in a hypertensive adult.

Little has been said about symptomatic vascular anomalies which are usually encountered in infants and small children. Only if routine roentgenograms are of excellent quality can they be suspected from evidence of abnormal compression of the trachea. Their presence can be confirmed by barium swallow and by tracheograms. For excellent summaries of these conditions the reader is referred to articles

such as those by Gross and Neuhauser,[26] and by Edwards.[20] "It would require a presentation of monographic size to list and describe all the anomalies which have been encountered."[26]

Aneurysm of the Sinus of Valsalva. Aneurysm of the sinus of Valsalva or of an aortic sinus may be large enough to project beyond the border of the heart, either on the right or on the left, and may appear to be a mediastinal mass (for example, Figures 5 and 6 in the article by Ostrum, Robinson, Nichols and Widmann[50]). Although we have seen several patients with aneurysms of the aortic sinuses, we have not encountered a case in which the lesion simulated a mediastinal mass.

Aneurysms of the Thoracic Aorta. Acquired thoracic aortic aneurysms usually do not present a problem in identification but occasionally they may simulate a mediastinal tumor (Fig. 21, *a*, *b*, *c* and *d*). Oblique and lateral roentgenographic views will usually clarify the diagnosis (Fig. 22, *a* and *b*), but angiocardiography or retrograde aortography may be required for

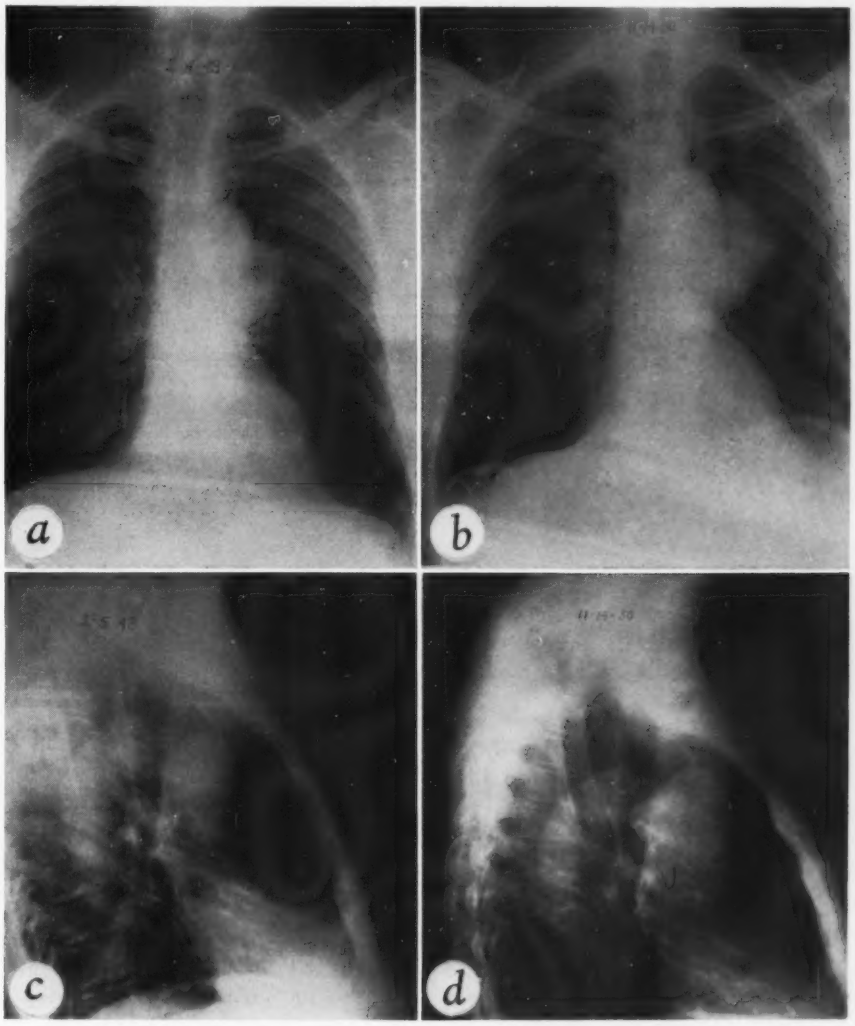


Fig. 21. Posteroanterior roentgenograms (*a* and *b*) and lateral roentgenograms (*c* and *d*) taken almost three years apart, depicting a growing aneurysm of the ascending thoracic aorta. It is obvious that this could easily be mistaken for a hilar mass. Note how the inferior aspect of the "mass" blends imperceptibly with the cardio-aortic shadow.

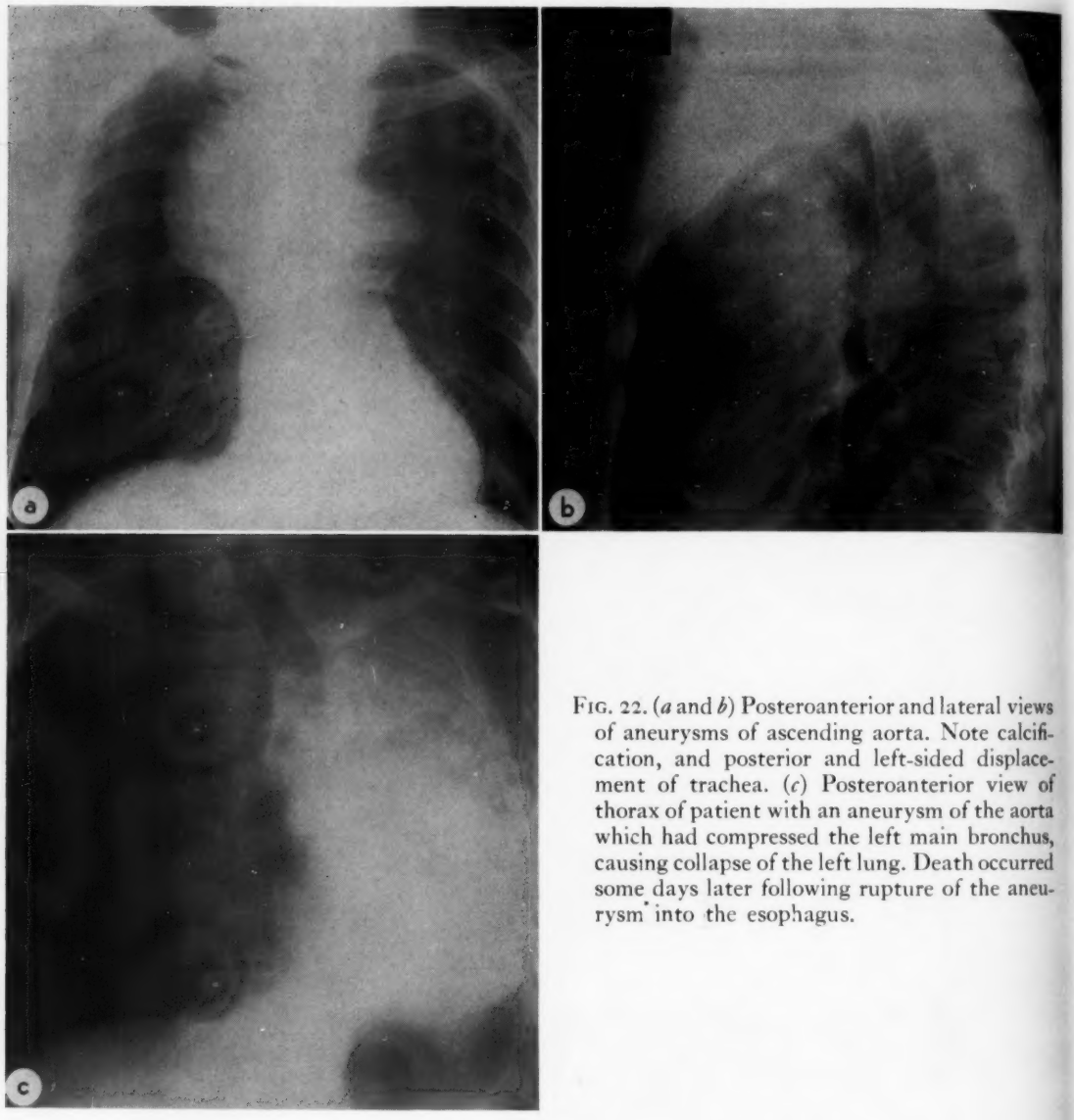


FIG. 22. (*a* and *b*) Posteroanterior and lateral views of aneurysms of ascending aorta. Note calcification, and posterior and left-sided displacement of trachea. (*c*) Posteroanterior view of thorax of patient with an aneurysm of the aorta which had compressed the left main bronchus, causing collapse of the left lung. Death occurred some days later following rupture of the aneurysm into the esophagus.

accurate identification. Of interest are aneurysms that may be large enough to cause symptoms and signs of bronchial obstruction (Fig. 22*c*), obstruction of a pulmonary artery, dysphagia, hoarseness or diaphragmatic paralysis.

Traumatic mediastinal aneurysms resulting from injury to the thoracic aorta or one of its major branches may appear identical to mediastinal neoplasms. A diagnosis of Hodgkin's disease was not far-fetched in the patient whose roentgenograms are presented in Figure 23*a*. Even though it was known that the patient had been in a severe automobile accident, lymphoma was thought to be the most likely diagnosis because of the roentgenographic appearance of the lesion and the facts that pulses in the extremities were normal and no murmurs were present. Exploration revealed that the innominate artery had been traumatically severed at its origin from the aorta, and that the mediastinal mass was a huge false aneurysm.

Congenital aneurysms of the descending portion of the aortic arch are rare. They

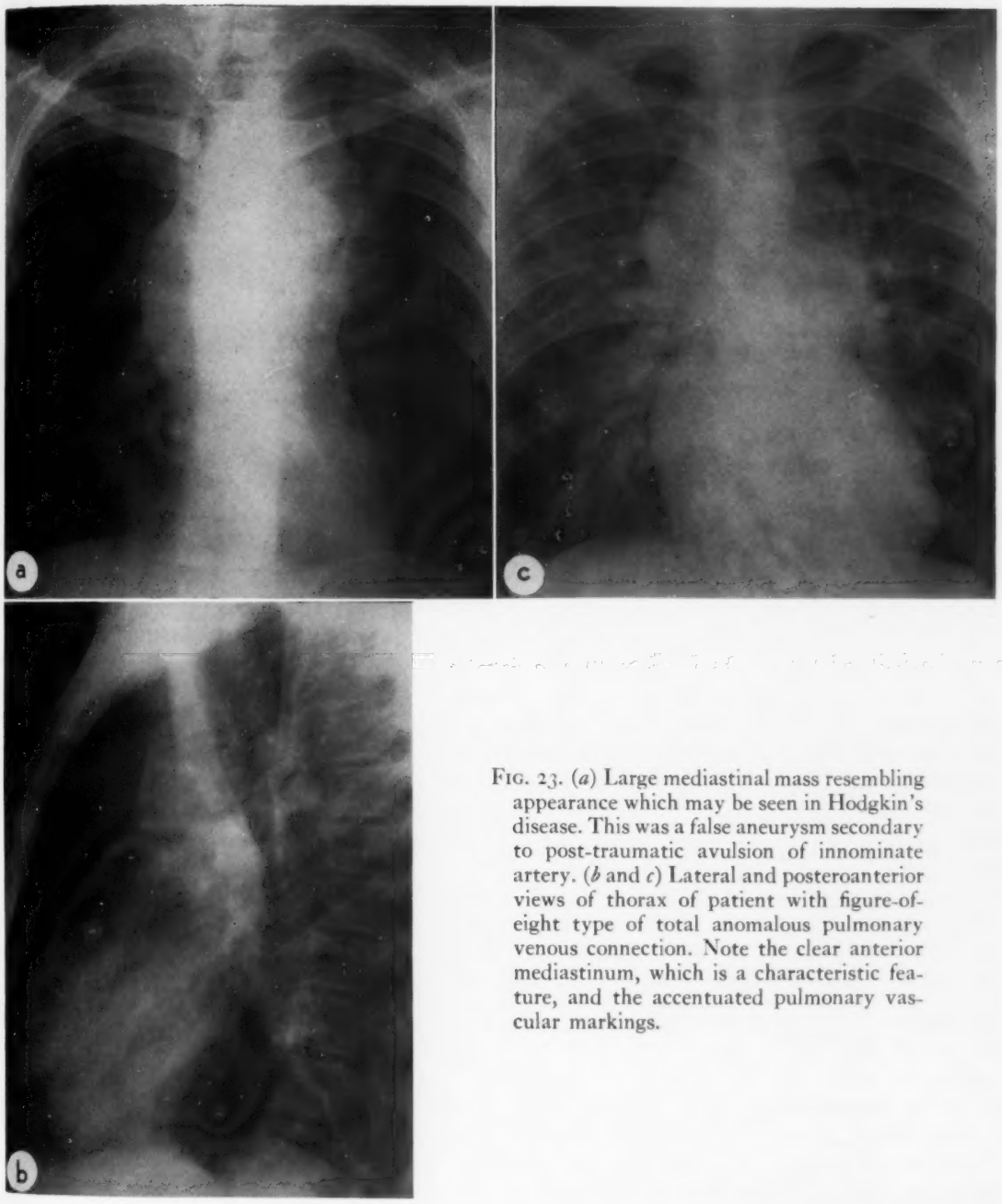


Fig. 23. (*a*) Large mediastinal mass resembling appearance which may be seen in Hodgkin's disease. This was a false aneurysm secondary to post-traumatic avulsion of innominate artery. (*b* and *c*) Lateral and posteroanterior views of thorax of patient with figure-of-eight type of total anomalous pulmonary venous connection. Note the clear anterior mediastinum, which is a characteristic feature, and the accentuated pulmonary vascular markings.

occur in the region of insertion of the ligamentum arteriosum.[58] Usually they are small (about the size of a hen's egg) and have some calcification in the periphery (Fig. 24, *a* and *b*). These lesions are easily diagnosed once one has become familiar with them, but it is quite possible to mistake them for a partially calcified posterior mediastinal tumor. Lateral and oblique views as well as fluoroscopy should facilitate diagnosis, but angiocardiography may be required for a definitive answer.

Aneurysm and Buckling of Innominate and Carotid Arteries. An aneurysm of the innominate artery may cause a shadow on the roentgenogram suggestive of a mass in

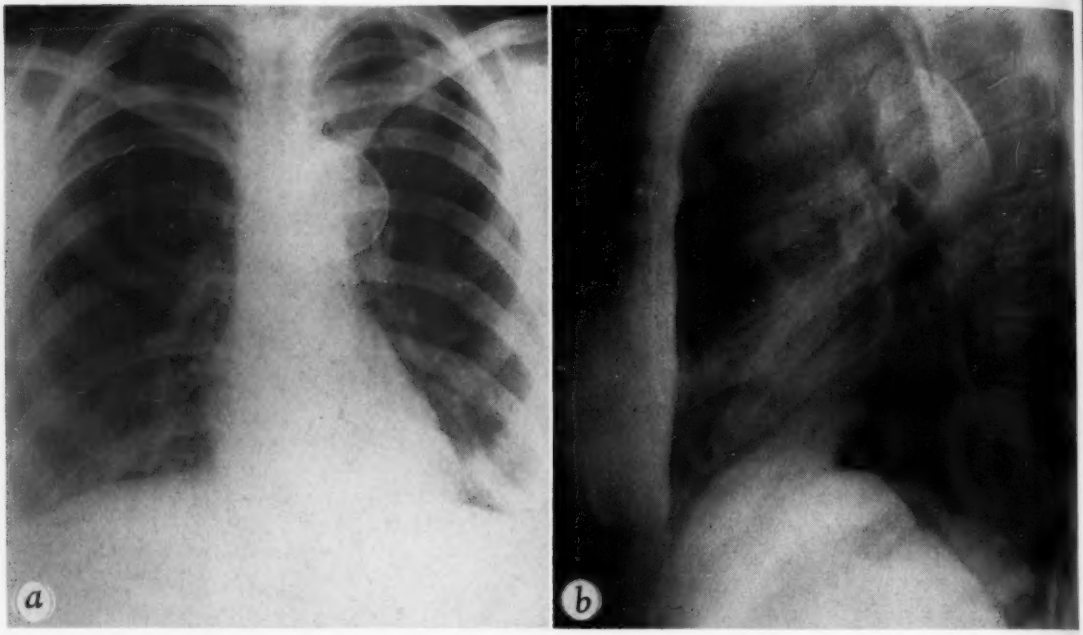


FIG. 24. (*a* and *b*) Posteroanterior and lateral views of thorax of a twenty-one year old woman with a congenital aneurysm of the aorta in the region of the ligamentum arteriosum. This is the typical site for these aneurysms.

the upper part of the mediastinum. Usually the aneurysm will be palpable and the diagnosis obvious. The walls of such aneurysms may contain calcium which may cause erosion of the medial aspect of the right clavicle (Fig. 16). Bone erosion of this type favors the diagnosis of aneurysm rather than of neoplasm.

A tortuous innominate or carotid artery may simulate a mediastinal mass, and an angiocardiogram may be required to differentiate it from an aneurysm.[34,35,43] The condition is benign and surgical treatment is not indicated.

Anomalous Right Subclavian Artery and Dysphagia Lusoria. In about 1 per cent of patients seen for roentgenologic investigation of the upper portion of the gastrointestinal tract, there is evidence of an anomalous course of the right subclavian artery. This vessel is the last branch to arise from the aortic arch, and in order to reach the right arm it has to pass either behind the esophagus or between the trachea and the esophagus. Its presence is easily detected during a barium swallow (Fig. 25, *a* and *b*).

Occasionally, the presence of the abnormal artery can be suspected on lateral roentgenographic views of the thorax by the anterior deviation of the tracheal shadow about level with the aortic arch (Fig. 25*c*). Rarely the anomaly is associated with dysphagia, and according to Asherson,[2] the term "dysphagia lusoria" was first applied in this relationship by Bayford in 1794.[4] It is remarkable that in many patients who have marked deformity of the esophagus caused by the anomalous vessel, no related symptoms are evident.

Compression of Esophagus by Descending Thoracic Aorta. Occasionally, in middle-aged and elderly patients with tortuosity of the descending thoracic aorta, the aorta will cause a varying degree of pressure on the esophagus. The level of this compression is usually in the lower third of the esophagus, and the deformity may be so marked as to cause complete, temporary holdup of the barium column. We have

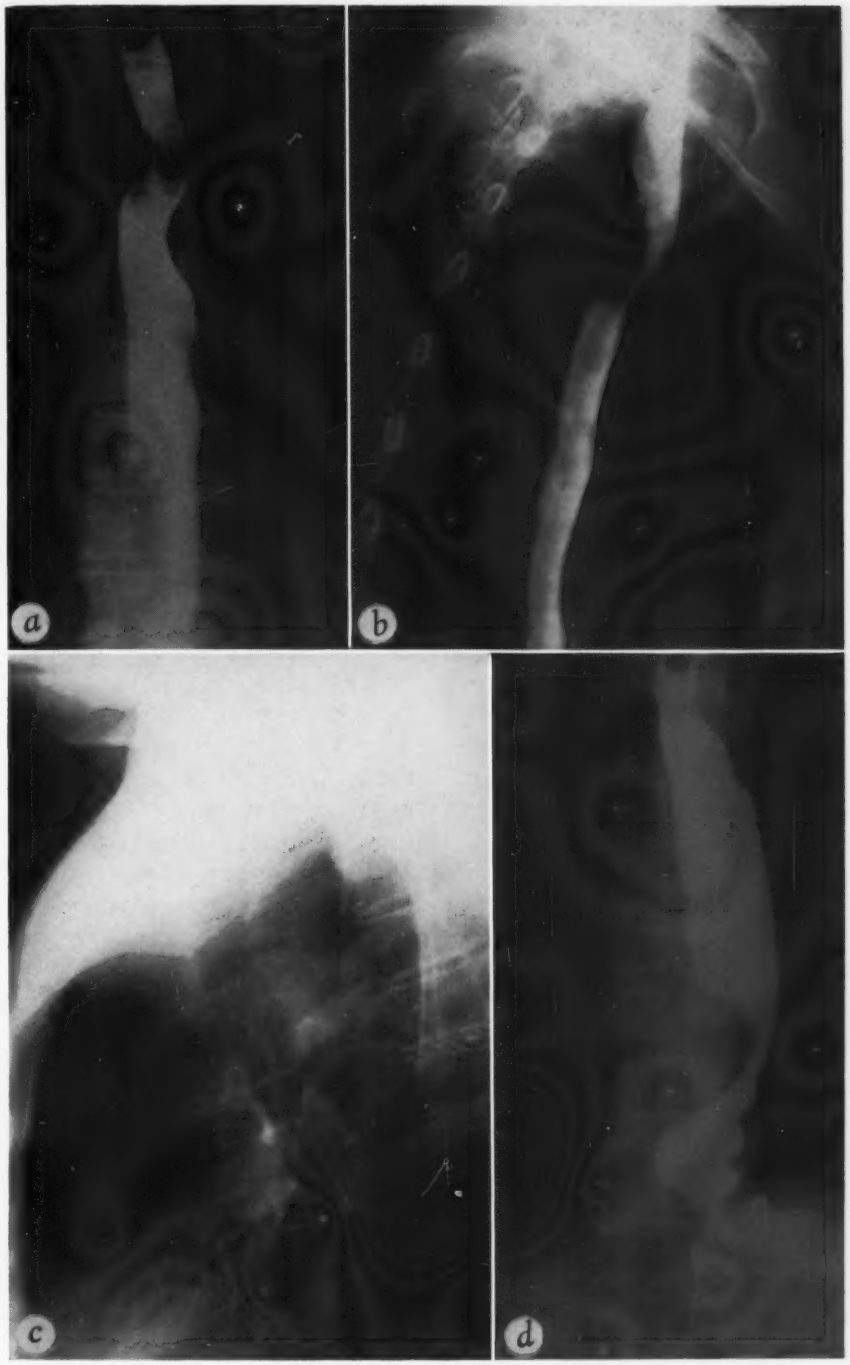


FIG. 25. (*a* and *b*) Anteroposterior and lateral projections of barium swallow showing oblique defect due to pressure by anomalous right subclavian artery. (*c*) Lateral view of thorax of patient who had had a carcinoma of the ovary removed several years before. Note localized anterior displacement of the trachea. Barium swallow showed typical defect of anomalous right subclavian artery on the esophagus at the same level as the tracheal deformity. (*d*) Barium swallow (anteroposterior projection) in a seventy-six year old woman, showing marked compression of the lower portion of esophagus by a tortuous and dilated descending thoracic aorta. Note the curved shadow of aorta as it passes behind the esophagus.

noted that when this happens, if the patient leans forward slightly, the pressure on the esophagus may be relieved. This deformity is occasionally associated with dysphagia, and we have seen a deformity so marked as to have been suspected prior to the patient's consultation at the clinic of having been due to a neoplasm (Fig. 25*d*). Mucklow and Smith[46] have been impressed by the following factors: "1. The variability of the point at which the aorta crosses behind the lower esophagus, and the difference of angulation at the crossing. 2. The variability in the degree of anterior displacement of the lower third of the esophagus by the distal thoracic aorta. 3. The occasional incidence of dysphagia *apparently* solely referable to compression of the esophagus by the aorta."

Conditions Affecting the Pulmonary Artery. Pulmonic Stenosis. Although the clinical diagnosis of pulmonic stenosis (without ventricular septal defect) does not usually cause much difficulty, occasionally the degree of poststenotic dilatation will be such as to arouse serious suspicion of a mediastinal tumor[9] (Fig. 26*a*). Clinically the picture may be complicated by the knowledge that a mediastinal mass compressing the main pulmonary artery may result in a pulmonary systolic murmur. It is obvious that in order to differentiate this condition from a left hilar tumor, specialized procedures such as cardiac catheterization or angiocardiography may be required. Fluoroscopy is not to be relied on in these cases, because even marked pulsation of the left hilar density may be due to transmitted pulsation of a tumor.[75]

Absence of a Pulmonary Artery. Several reports have dealt with absence of a pulmonary artery in the presence of a lung that is normally connected to the bronchial tree.[23,69] The lung on the side where the pulmonary artery is absent is smaller than the opposite lung, and the hemithorax in which it lies is smaller than the opposite hemithorax (Fig. 26*b*). Usually the normally connected lung is seen, on the routine roentgenogram, to herniate across the midline, displacing the mediastinal structures to the opposite side. Normal vascular markings are not seen on the side of the absent pulmonary artery, but the lace-like pattern of bronchial arteries may be identified in the region of the hilus.[77] An angiographic study will demonstrate the defect strikingly (Fig. 26*c*). Emanuel and Pattinson[23] have made the interesting observation that absence of the left pulmonary artery is almost invariably accompanied by the tetralogy of Fallot. They stated, "In cases where the heart was normal or the cardiac anomaly did not involve the bulbus cordis, it was nearly always the right pulmonary artery that was absent."

Aneurysm of the Pulmonary Artery. Up to 1947, only 147 authentic cases of aneurysm of the pulmonary artery (Fig. 26*d*) had been reported.[16] "Idiopathic or primary dilatation of the pulmonary artery is very rare. It can be diagnosed only after all other causes of pulmonary artery dilatation, such as pulmonic stenosis, congenital heart disease (interatrial and interventricular septal defects and patent ductus arteriosus), rheumatic heart disease, cor pulmonale, heart failure and syphilis, are excluded."[70] It should be stressed, however, that angiocardiography may be mandatory in order to be certain of the nature of the structure.

It has been demonstrated that fluoroscopy is inferior to the impression gained from the posteroanterior views on routine roentgenograms in the evaluation of the smoothly-rounded left hilar "mass," and that angiocardiography may be the only certain way of identifying the nature of such a structure.[75]

Anomalous Course of Left Pulmonary Artery to Right of Trachea. Welsh[76] and Potts[53] and their co-workers have described a curious anomaly of the left pulmonary artery which may be associated with respiratory difficulties in infancy. In this anomalous development the left pulmonary artery passes anteriorly and to the right of the lower portion of the trachea and over the right main bronchus before passing behind the trachea to the left lung.

Conditions Affecting the Thoracic Veins.

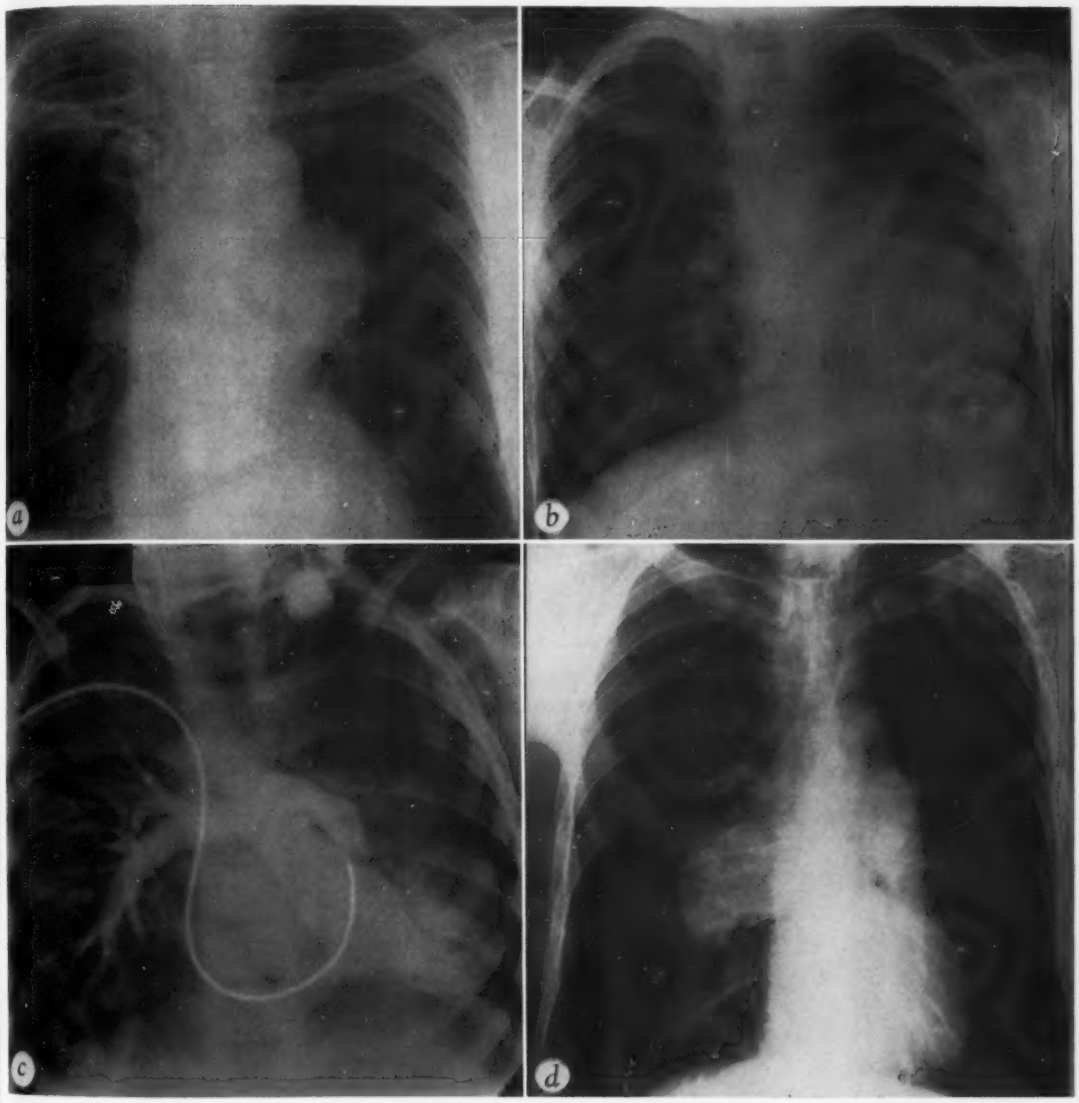


Fig. 26. (*a*) Valvular pulmonic stenosis (without ventricular septal defect) and marked poststenotic dilatation of pulmonary trunk. (*b* and *c*) Agenesis of the left pulmonary artery associated with tetralogy of Fallot. (*b*) Note that the left hemithorax is small, the left diaphragm is elevated and the heart is shifted to the left. Poor lung-markings on left. (*c*) Selective angiogram showing absence of pulmonary arterial supply to left lung. (*d*) Posteroanterior view of aneurysm of right pulmonary artery. Note calcification in its walls.

Anomalous venous connection with the right lung has already been discussed under Pulmonary Entities.

Total Anomalous Pulmonary Venous Connection. On a roentgenogram the type of total anomalous venous connection referred to as the figure-of-eight[66] could be mistaken for a mediastinal mass.

In this type of connection the right lung drains its venous blood across the midline to the left where it joins the venous blood from the left lung in the so-called left vertical vein or partially persistent left superior vena cava. This large vein causes the widening of the mediastinal shadow to the left. The blood drains from this vein into the left innominate vein and thence to the normal superior vena cava, which becomes enlarged and causes widening of the mediastinal shadow to the right. This appearance may give rise to a mistaken diagnosis of mediastinal mass. However, the lung

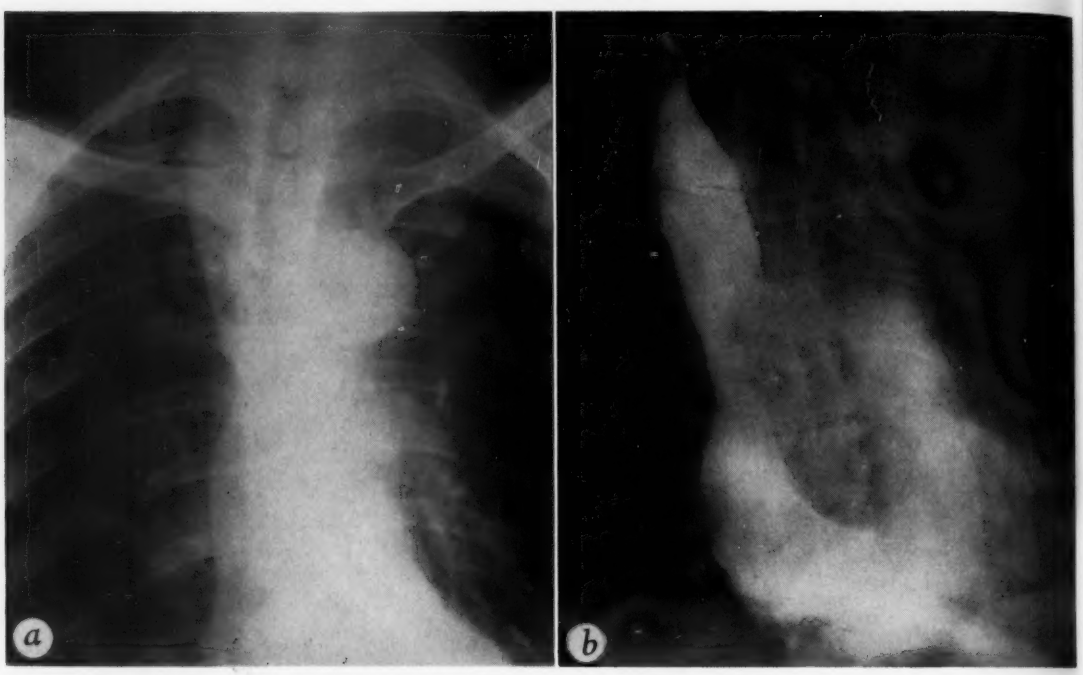


FIG. 27. (*a*) Posteroanterior view showing mediastinal shadow widened toward the right. (*b*) Angiocardiogram shows shadow is the superior vena cava.

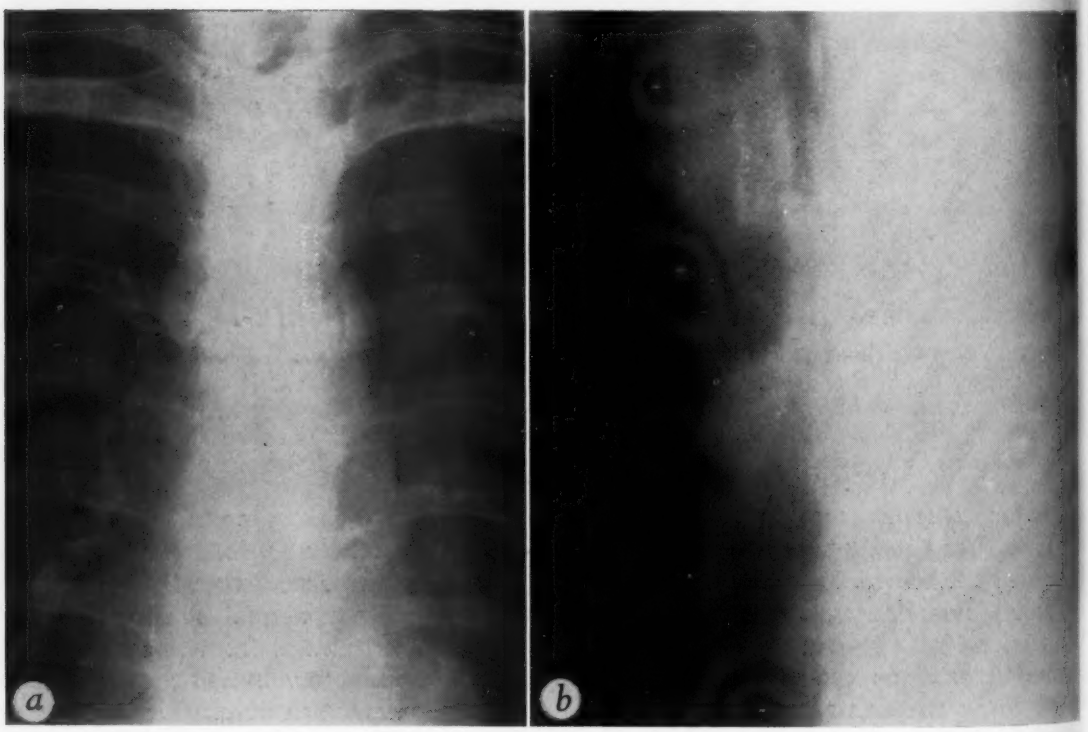


FIG. 28. (*a*) Posteroanterior view of thorax showing prominent shadow of azygos vein to the right of the lower trachea. (*b*) Laminagraphic view of a normal azygos vein, just before it enters the superior vena cava.

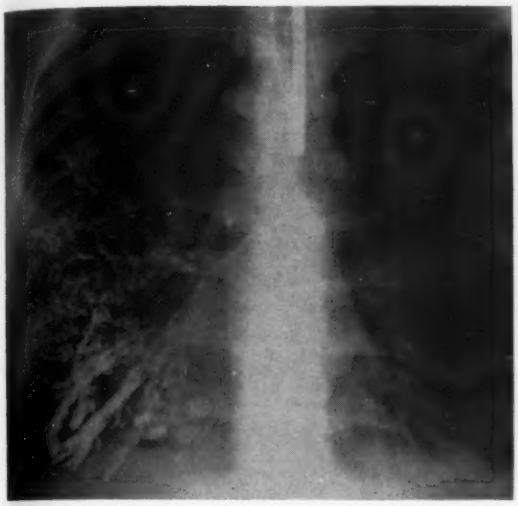


Fig. 29. Dextrocardia and bronchiectasis.

fields will usually show the typical findings of pulmonary recirculation, and on the lateral view the anterior mediastinum is characteristically clear[7] (Fig. 23, *b* and *c*).

Superior Vena Cava. Not infrequently the shadow of the right edge of the superior vena cava will be visible on the routine thoracic roentgenogram, and occasionally its appearance will suggest superior mediastinal widening. On occasion one may have to resort to a venogram to prove whether or not such a shadow is, in fact, that of the superior vena cava (Fig. 27, *a* and *b*).

When the superior vena cava is situated on the left, it may cause a confusing shadow suggestive of abnormal mediastinal widening to the left of the aortic arch. In such a case a venogram, through the left arm, also may be required to prove the nature of the shadow. Persistent left superior vena cava may be associated with a congenital heart lesion, and the passage of an intracardiac catheter via the left arm not infrequently

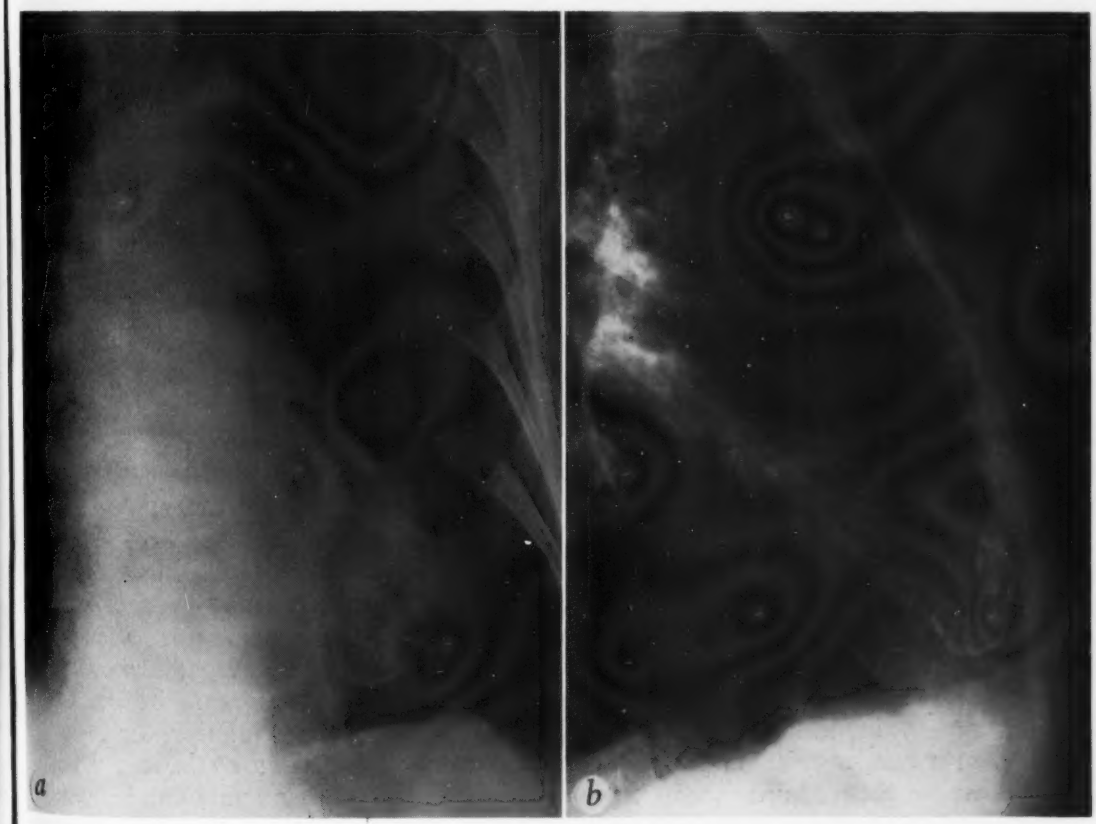


Fig. 30. (*a* and *b*) Posteroanterior and lateral views of thorax of patient who had suffered a transmural myocardial infarct. Note the calcification in the myocardial scar.

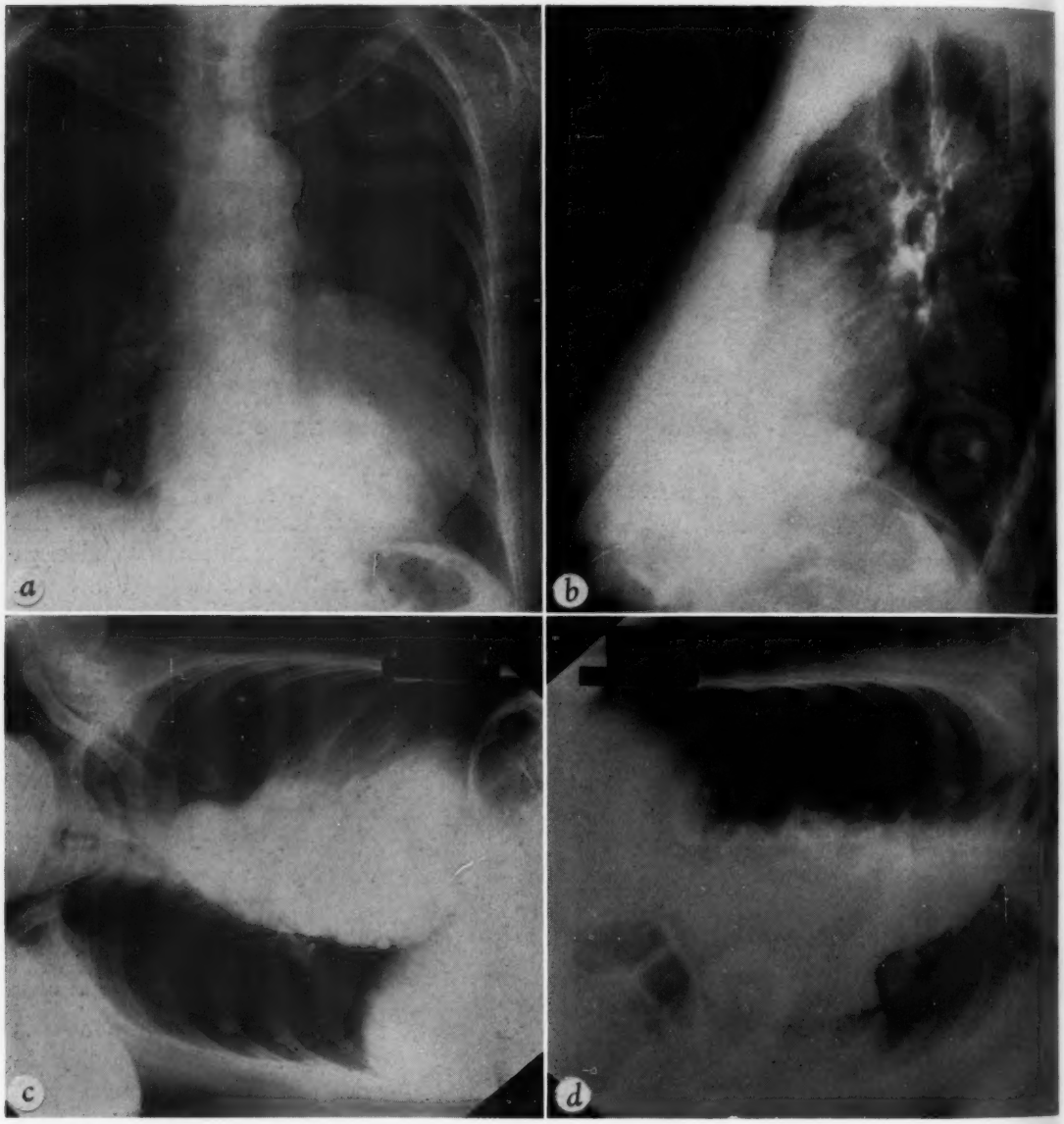


FIG. 31. (*a* and *b*) Posteroanterior and lateral views of large pericardial cyst. (*c* and *d*) Right and left lateral decubital views showing marked alteration in shape of the "springwater" containing cyst.

uncovers the presence of a left superior vena cava.

Azygos Vein. The roentgenographic picture presented by the anomalous course of the azygos vein is familiar. Of more interest are those cases in which the normally placed azygos vein casts a shadow in the angle of the trachea and the right main bronchus which may be mistaken for a pathologic mass (Fig. 28, *a* and *b*).[22] Cases also have been described in which an aneurysmal malformation of the azygos vein may give the appearance of a paratracheal tumor,[60] and enlargement of the shadow of the azygos vein is seen in cases of obstruction of the inferior vena cava.[5] In such cases the azygos vein is the pathway for the blood that would normally have returned to the heart via the inferior vena cava.

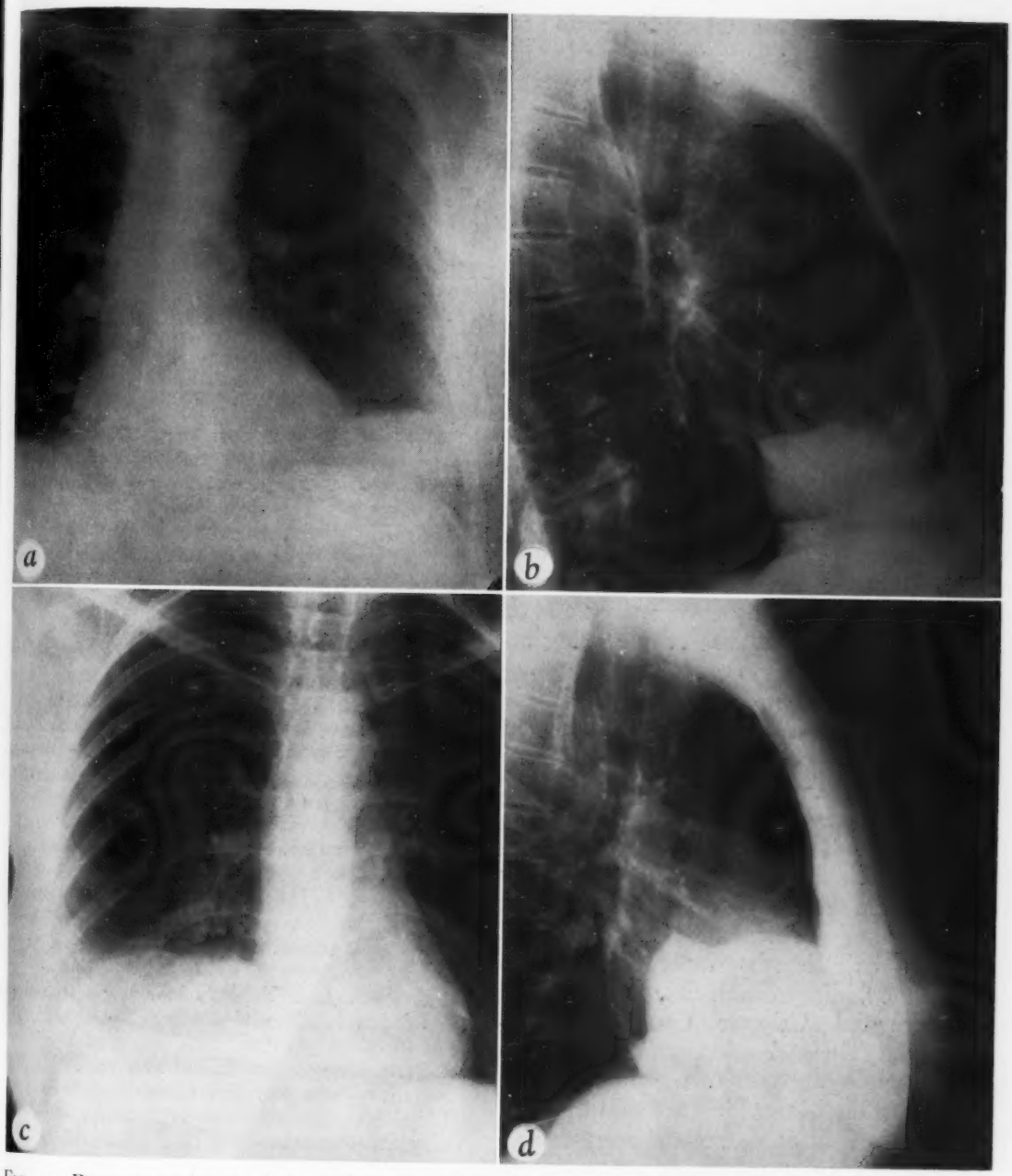


Fig. 32. Posteroanterior and lateral views of pericardial cyst (*a* and *b*). The peaking toward the oblique fissure on the lateral view is a characteristic feature of some pericardial cysts in this region, as can be seen from the roentgenograms of another case (*c* and *d*).

Conditions Affecting the Heart and Pericardium. Kartagener's Triad—Dextrocardia, Bronchiectasis and Sinusitis (Fig. 29). The association of dextrocardia or situs inversus with bronchiectasis was first established by Kartagener.[37] Olsen[49] examined the records of 85 patients with dextrocardia who had been seen at the Mayo Clinic and found an incidence of bronchiectasis of 16.5 per cent as opposed to about 1 per cent in the general patient population at the clinic. It is thought that there is

probably a developmental predisposition to bronchiectasis in patients with inversion of the viscera.

Calcified Myocardial Infarcts. Several articles dealing with myocardial calcification following coronary thrombosis and myocardial infarction have appeared in the literature. In a large percentage of these cases a striking similarity of the roentgenographic appearance of the myocardial calcification has been manifested. However, unfamiliarity with the roentgen picture may be the cause of some diagnostic confusion, and such conditions as calcified intrathoracic cysts may be considered. Fluoroscopy or oblique and lateral roentgenograms are required to demonstrate the cardiac location of the calcium. Hankamp[29] has recently presented a typical case and has included a brief review of the literature. We have recently encountered 2 such cases, the roentgenogram of one being reproduced in Figure 30, *a* and *b*.

Ventricular Aneurysm. It is conceivable that a ventricular aneurysm following myocardial infarction may simulate a mass adjacent to the apex of the heart (Fig. 11, *c* and *d*). However, the fact that the shadow cast by such an aneurysm blends homogeneously with the remainder of the cardiac shadow should be sufficient evidence that it is part of the heart. Furthermore, the characteristic electrocardiographic and fluoroscopic findings clinch the diagnosis.

Pericardial Celomic Cysts and Diverticula. These developmental anomalies can occur in any part of the mediastinum related to the heart. They are said to result from failure of one of the primitive pericardial lacunae to fuse with others during embryonic formation of the pericardial sac.[41] They may retain a small communication with the main pericardial cavity (pericardial diverticulum) or they may be quite separate from the pericardial lumen (pericardial celomic cyst, "springwater" cyst). Owing to the fact that they are thin walled, they frequently will show considerable change in shape when studied in the right and the left lateral decubitus positions (Fig. 31, *a*, *b*, *c* and *d*). This allows considerable accuracy in roentgenographic diagnosis. When they present in the cardiophrenic region, the lateral view not infrequently shows a characteristic peaked appearance (Fig. 32). Occasionally, pericardial celomic cysts will present a fantastic roentgenographic appearance, as was the case, for instance, in the one presented by Gerbasi.[25]

Mayo Clinic
Rochester, Minnesota

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OCCLUSION OF THE ABDOMINAL AORTA*

By SERGIO CHIAPPA
MILANO, ITALY

SINCE the introduction of aortography in common roentgenologic practice, partial or total occlusion of the abdominal aorta, which up to now had nearly always been a postmortem finding, has been frequently diagnosed in living patients and successfully operated upon.

The two pathologic conditions which may cause an occlusion of the aorta are acute embolism and thrombosis. The embolus is usually located at the level of the aortic bifurcation or in one of the two iliac arteries, while the thrombosis, under certain conditions, leads to high occlusion of the abdominal aorta. In such cases we are confronted with thrombi originating from one of the iliac arteries or from the aortic "carrefour," which produces an occlusion of the contralateral iliac artery. In this way, a block is formed at the level of the aortic bifurcation, which leads to an extension of the thrombosis to a higher level, reaching in some instances the region of the renal arteries. At autopsy, usually an organized thrombus is found which is completely adherent to the aortic wall.

The lesions which most frequently lead to the formation of this type of thrombosis are atherosclerosis and obliterating arteritis due to alterations, which appear after the age of fifty in hypercholesterinemic subjects and in heavy smokers.

The early clinical symptoms have their onset in the first stage of the disease, the so-called compensated obliteration of Leriche, but they are not distinctive in character. Thus, we may observe a reduced endurance to walking, accompanied by paleness of the legs and feet, which may show up even when the patient is only standing. Less frequently, in more advanced cases, other signs may become manifest such as dystrophy of the skin and nails, and atrophy of both lower extremities. Clinical investigation at this time often discloses complete absence of pulse in the dorsal artery of the foot, in the posterior tibial artery, and in the popliteal, femoral and iliac arteries.

The symptoms of the second stage, which is called decompensated obliteration, are more characteristic. They consist of cyanosis of both legs and feet, accompanied by small ulcerations, situated at pressure points. Soon gangrene sets in, affecting one or—in some cases—both legs, associated with severe pain. Frequently, a descending progression of the thrombus with an ensuing peripheral thrombosis may be observed.

Experience shows that abdominal aortography, performed by direct puncture, usually permits a clear-cut diagnosis of the nature and seat of the alteration in occlusion of the abdominal aorta. In some patients presenting complaints of tiredness of the lower extremities with intermittent claudication, it may show parietal involvement with only partial occlusion. When, however, thrombosis has reached an advanced stage and the occlusion is complete, aortography will readily demonstrate the precise location and extent of the process as well as the status of the collateral circulation.

MATERIAL AND TECHNIQUE

This study is based on 5 cases (out of a total of 104 cases) in which abdominal aortography was performed at the I Radiologic Department, Söder Hospital, Stockholm, Sweden, and at the Institute of Radiology of the University of Pavia, Italy.

In all cases aortography was performed by direct puncture of the abdominal aorta just above the level of the renal arteries. Forty to 50 cc. of contrast medium (um-

* From the Institute of Medical Radiology and Physical Therapy (Director: Prof. A. Ratti), University of Milano, Italy.

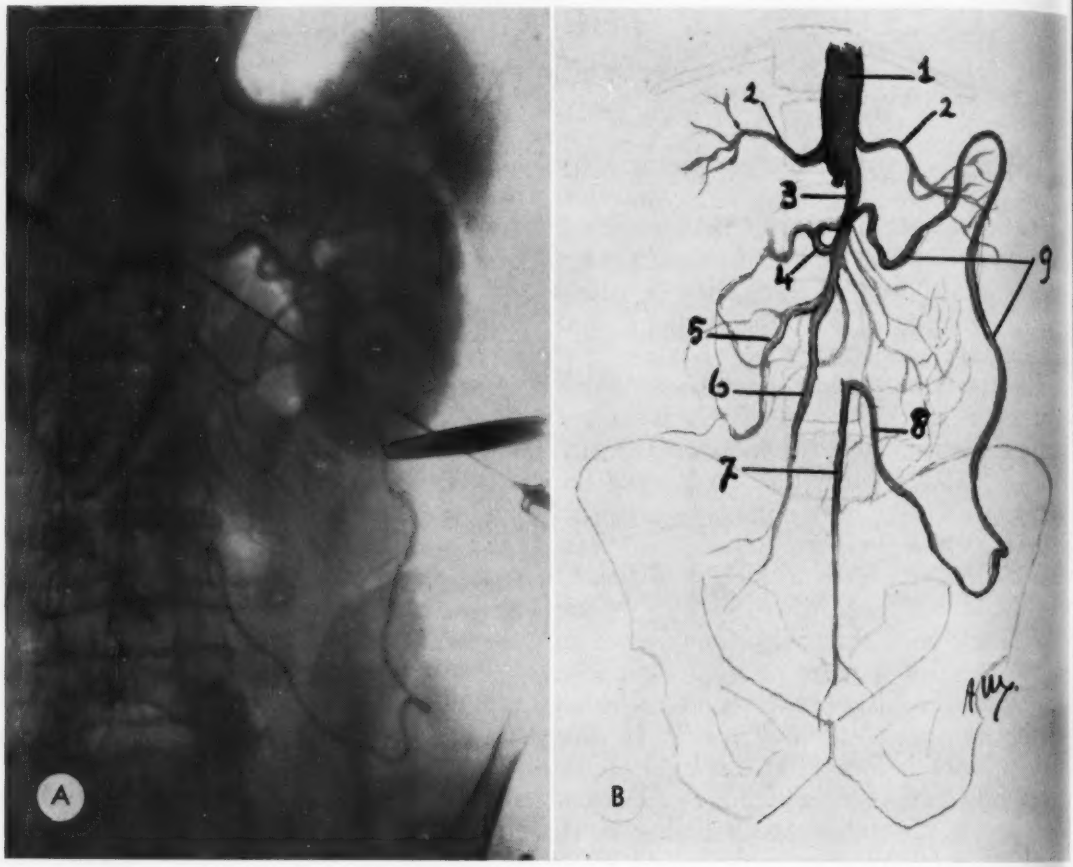

Fig. 1. Case 1. (*A*) Aortogram showing complete occlusion of the distal abdominal aorta. (*B*) Tracing of *A*. (1) Aorta; (2) renal artery; (3) superior mesenteric artery; (4) middle colic artery; (5) right colic artery; (6) ileocolic artery; (7) inferior mesenteric artery; (8) left colic artery; (9) Riolan's anastomotic arch.

bradil) at a concentration of 50 or 70 per cent was injected within three to six seconds.

REPORT OF CASES

Case 1. A.E., a male aged fifty-eight. The patient had complained for some years of pain of the lower extremities, accompanied by cramps of the left leg, which had become more intense during the last few months. A few days before being examined, the patient suffered an acute attack of pain in the left leg and a feeling of coldness in both legs. No pulse could be felt over the femoral arteries.

Aortography was performed through direct puncture of the aorta just above the origin of the renal arteries with injection of 50 cc. umbradil 50 per cent in about three seconds. The distal part of the abdominal aorta was completely occluded. There was a gradual narrowing of the lumen at the site of the occlusion, giving a cone-shaped appearance. The opaque medium passed into the pelvis through the collateral pathways. The iliac arteries could not be visualized (Fig. 1, *A* and *B*).

Case 11. E.E., male aged forty-eight. The patient had complained for a long period of time of vascular disturbances, which were attributed to an obliterating arteriosclerosis. Three years before admission, the patient had undergone a Smithwick operation. At the time of his recent admission, he again had severe pain in both legs, more pronounced on the left side. No arterial pressure could be measured in the lower extremities.

Aortography was performed through direct puncture of the aorta at the level of the second lumbar vertebra on the left side with 45 cc. of umbradil 50 per cent, injected in about three seconds. The aortic caliber was considerably

reduced, its diameter being 2.3 mm. at the level of the intervertebral disk between the second and third lumbar vertebra. A complete block was observed at the level of the third lumbar vertebra so that the contrast medium passed below that level only through collateral pathways, which were more developed on the left side. Nonfilling of the great arteries of the small pelvis was noted (Fig. 2, *A* and *B*).

Case III. D.R., male aged fifty-two. The patient had been treated since 1950 for pulmonary tuberculosis. For a period of about twelve months, he had complained of cramps of the arterial type in the right leg. During the morning of the examination, he felt an acute severe pain in the left leg. The patient, who suffered from paresthesia of the gluteal region, said he felt like "sitting on a wooden bar." After this attack, he also noticed that both legs were cold, there was a marked reduction of the superficial and deep sensation and he was unable to raise his legs.

Aortography was performed through a direct puncture of the aorta at the level of the first lumbar vertebra with 40 cc. of umbradil 70 per cent being injected in five seconds. A block was noted at the level of the third lumbar vertebra. The aorta appeared to be dilated above this block, but the lower outlines of the opaque column were convex and regular (Fig. 3, *A* and *B*).

The patient had undergone a Smithwick operation and retrograde arteriography had been performed by introducing a small catheter into the right femoral artery. The femoral and iliac arteries on the same side had to be freed partially. The catheter was introduced without difficulty for some centimeters and two injections (one manually and the second under oxygen pressure) of 30 cc. umbradil 50 per cent were made. The contrast medium passed into the aorta through the unobstructed arteries and filled the distal part of the abdominal aorta. The thrombus was thus shown to occupy the

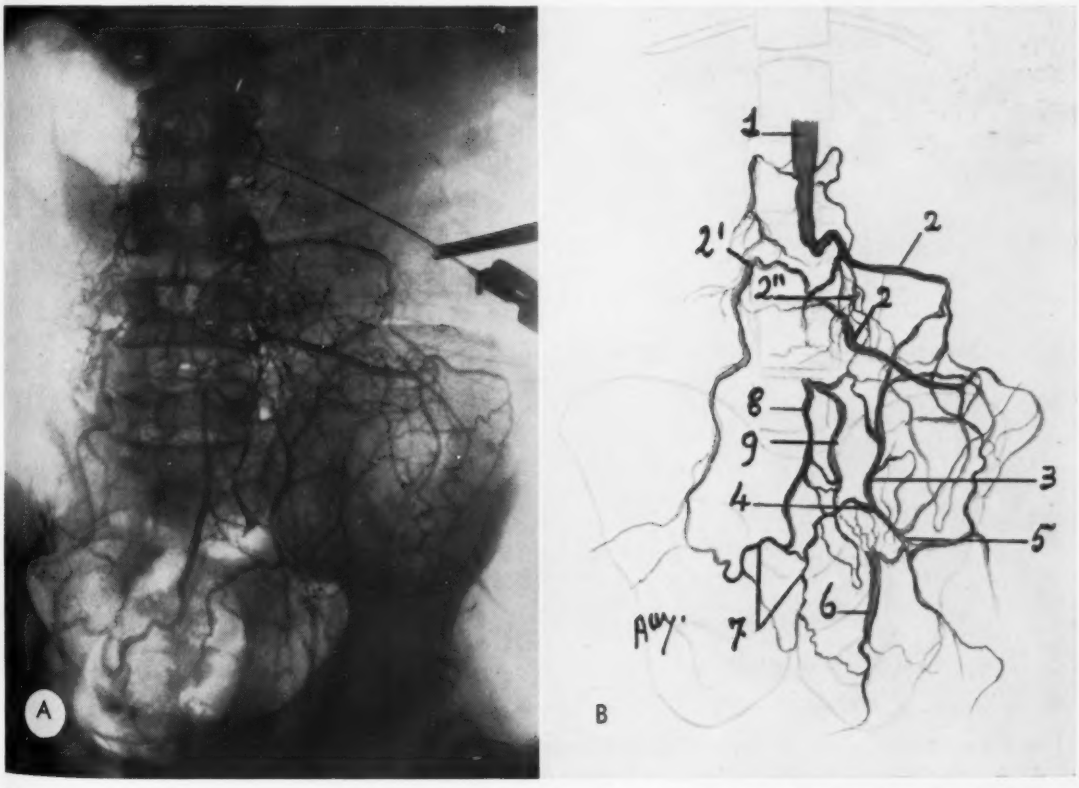

Fig. 2. Case II. (*A*) Aortogram showing complete occlusion of the aorta at L 3. (*B*) Tracing of *A*. (1) Aorta; (2–2') lumbar artery; (2'') vertebral circle; (3) iliolumbar artery; (4) lateral sacral artery; (5) gluteal artery; (6) internal pudendal artery; (7) hemorrhoidal artery; (8) inferior mesenteric artery; (9) left colic artery.

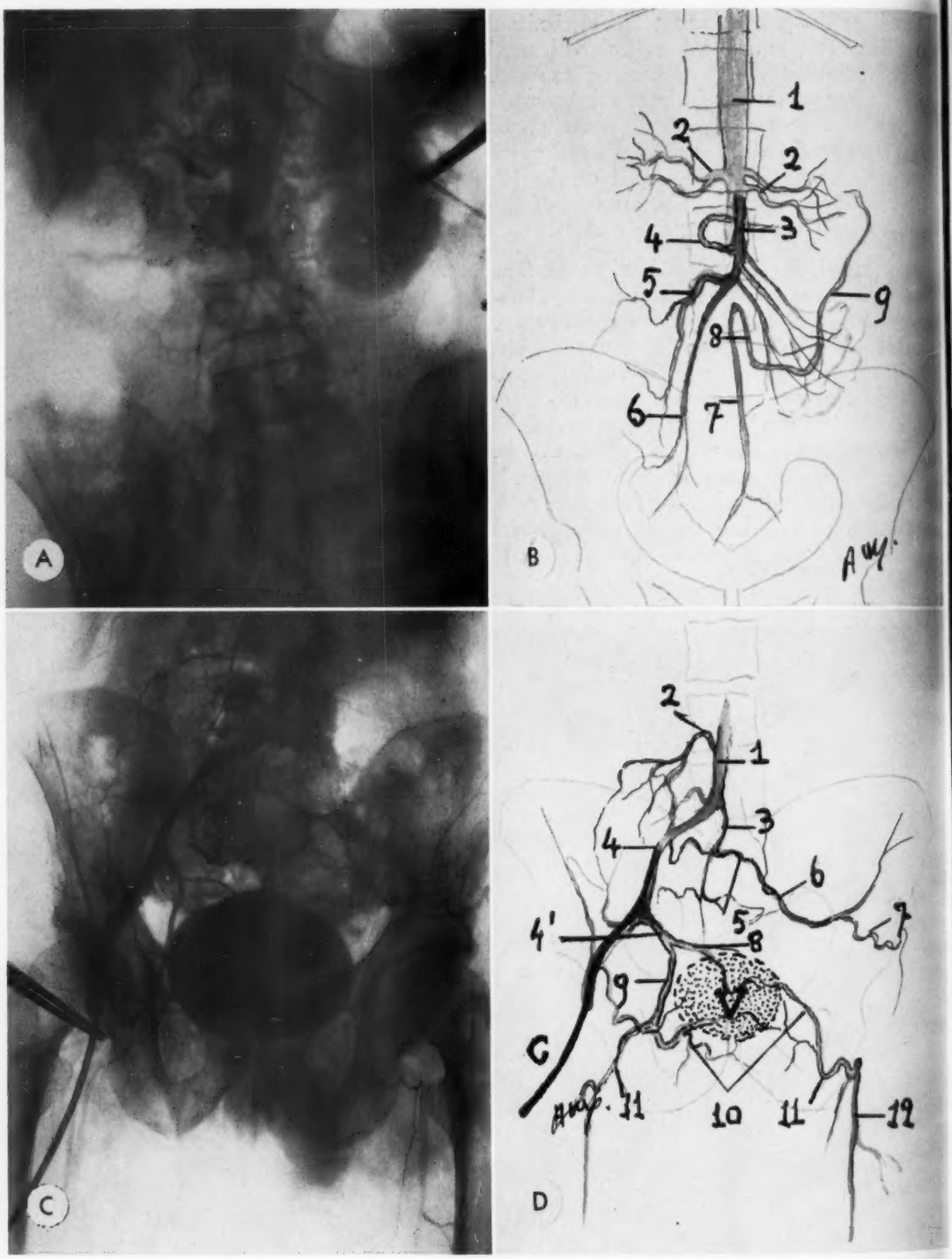

FIG. 3. Case III. (*A*) Aortogram showing complete occlusion of the aorta at L 3. (*B*) Tracing of *A*. (1) Aorta; (2) renal artery; (3) superior mesenteric artery; (4) middle colic artery; (5) right colic artery; (6) ileocolic artery; (7) inferior mesenteric artery; (8) left colic artery; (9) Riolan's anastomotic arch. (*C*) Retrograde right femoral arteriogram after Smithwick operation showing site of thrombus. (*D*) Tracing of *C*. (1) Aorta; (2) lumbar artery; (3) middle sacral artery; (4) common iliac artery; (4′) internal iliac artery; (5) segmental sacral artery; (6) lateral sacral artery; (7) gluteal artery; (8) vesical artery; (9) internal pudendal artery; (10) urogenital network; (11) obturator artery; (12) femoral artery.

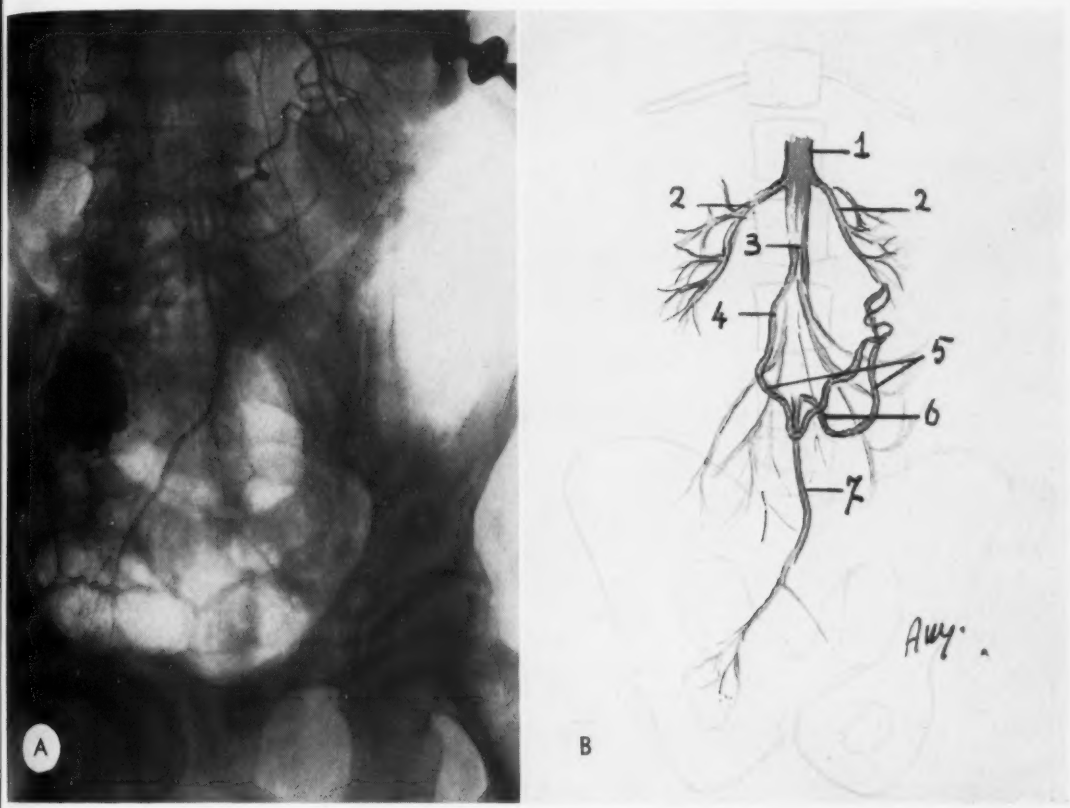

FIG. 4. CASE IV. (*A*) Aortogram showing complete occlusion of the aorta below the renal arteries. (*B*) Tracing of *A*. (1) Aorta; (2) renal artery; (3) superior mesenteric artery; (4) middle colic artery; (5) Riolan's anastomotic arch; (6) left colic artery; (7) inferior mesenteric artery.

entire lumen of the distal abdominal aorta, causing passage from the iliac artery into the femoral artery (Fig. 3, *C* and *D*).

CASE IV. N.E., male aged sixty-five. The patient was subjected to a Smithwick operation (third and fourth lumbar ganglia on the left side) in 1943. His general condition had improved after this surgical intervention.

Pain of the legs, though not the same as noticed ten years before, was again present in 1953. Since November, 1953 the pain became so severe that the patient could not walk. No pulse was felt over the femoral arteries.

Aortography through a direct puncture of the aorta at the level of the first lumbar vertebra was performed with 45 cc. of umbradil 70 per cent being injected. No filling of the abdominal aorta could be seen, but there was opacification of the renal arteries and of the widespread visceral collateral pathways (Fig. 4, *A* and *B*).

Twelve days after aortography, the patient died from heart failure and autopsy showed the presence of a long thrombus obliterating the aorta, which started just below the renal arteries, involved the iliac and femoral arteries, and reached the root of the thigh. The superior mesenteric artery was partially closed by a free thrombus.

CASE V. F.A., male aged seventy. The patient had been suffering for many years of intermittent pain of the legs which was quite severe in the last few months. Walking became difficult and the inferior extremities became easily tired. No pulse could be felt over the femoral arteries.

Aortography was performed with 40 cc. of umbradil 50 per cent being injected but no visualization of the abdominal aorta was obtained, though the femoral arteries, in a later film, showed a partial filling (Fig. 5, *A*, *B* and *C*).

At operation, a long thrombus was found in the distal part of the abdominal aorta, reaching the bifurcation and affecting the abdominal

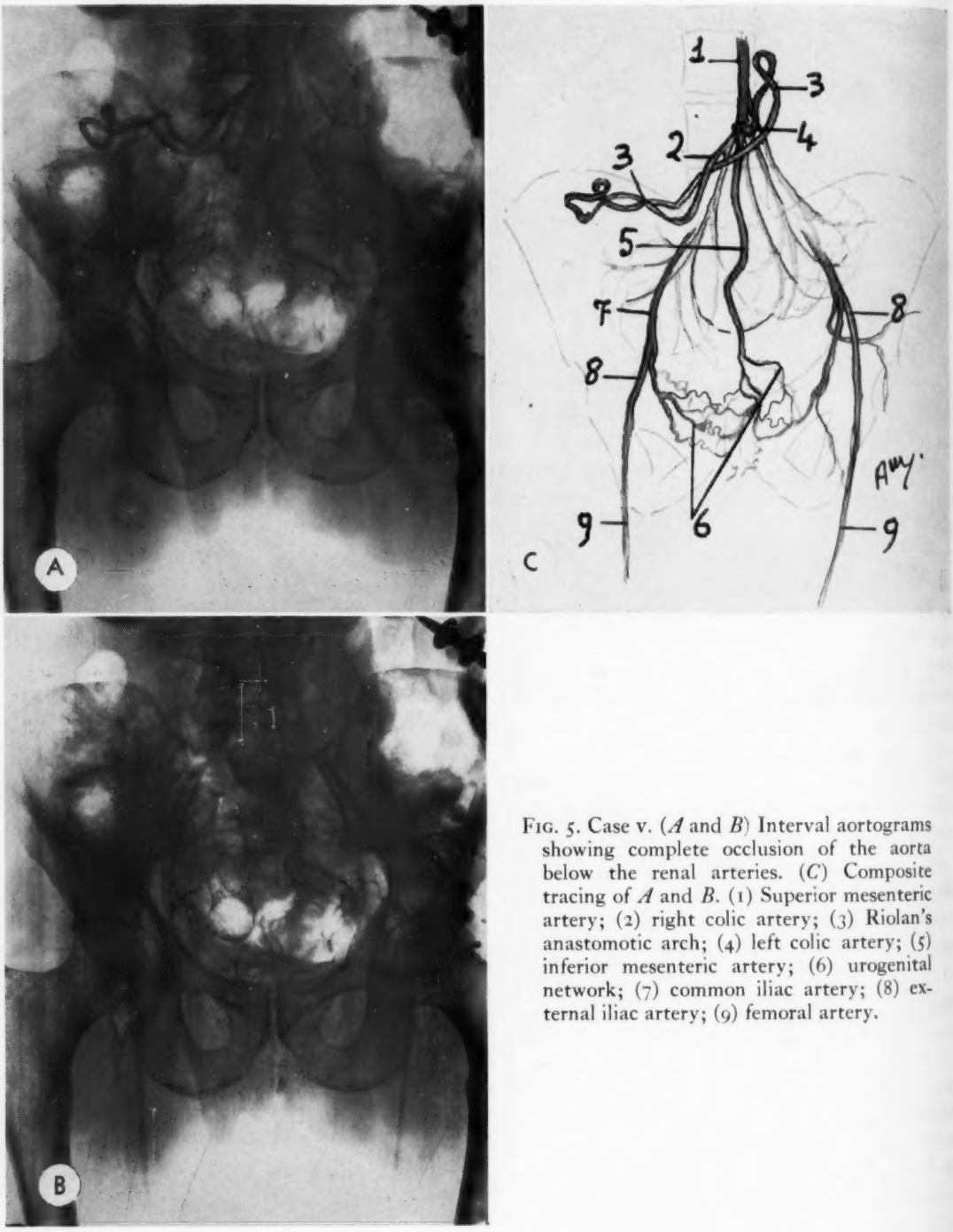

Fig. 5. Case v. (*A* and *B*) Interval aortograms showing complete occlusion of the aorta below the renal arteries. (*C*) Composite tracing of *A* and *B*. (1) Superior mesenteric artery; (2) right colic artery; (3) Riolan's anastomotic arch; (4) left colic artery; (5) inferior mesenteric artery; (6) urogenital network; (7) common iliac artery; (8) external iliac artery; (9) femoral artery.

portion of the iliac arteries. The thrombus spread upwards to a point below the origin of the renal arteries, partially closing the superior mesenteric artery.

DISCUSSION

As may be seen, it was possible in all of our cases to demonstrate by means of aortography high blocks of the abdominal aorta with complete occlusion of the lumen and accompanied by the formation of a well developed collateral circulation.

Of particular interest is the collateral

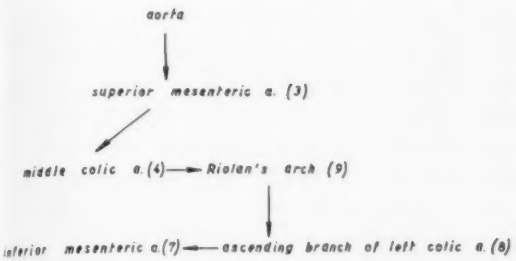

Fig. 6. Type of collateral circulation between the superior mesenteric artery and the inferior mesenteric artery (Case I).

circulation which has developed gradually, thereby enabling the patients to survive the otherwise fatal outcome of these severe obstructions of the abdominal aorta.

The first case (Fig. 1, *A* and *B*) is an example of a type of collateral circulation in which the superior mesenteric artery through its right side branches, *i.e.*, middle colic artery (4), right colic artery (5) and ileocolic artery (6), supplies the inferior mesenteric artery by way of the long arterial arch of Riolan (9). This arch is a pre-existing anastomosis between the ascending branch of the middle colic artery (4) and the corresponding branch of the left colic artery (8), the latter being a collateral branch of the inferior mesenteric artery (7) (Fig. 6).

In Case III (Fig. 3, *A–D*) similar, though less evident, findings, owing to the greater extension of the occlusion, were present. A good visualization was obtained of the territory supplied by the superior mesenteric artery (3) with its right side branches, *i.e.*, middle colic artery (4), right colic artery (5) and ileocolic artery (6), and the intestinal vessels. The supply of the inferior mesenteric artery (7) and its left colic branch (8) was made possible through the anastomotic Riolan's arch (9), which was not completely visible on the aortogram (Fig. 3, *A* and *B*). In this case, a Smithwick operation had been combined with the removal of a thrombus from the femoral artery, thus allowing the introduction of a catheter (Fig. 3, *C* and *D*) which was made to reach the right external iliac artery. In this manner, a retrograde injection had been obtained of the common iliac artery (4) and of the terminal part of the aorta (1). One could see now some of the right lumbar branches (2) of the aorta and the terminal middle sacral branch (3), with its segmental anastomosis with the lateral sacral artery of the opposite side (6). Through these collaterals, a filling of some of the left gluteal vessels (7) became possible. Part of the contrast medium, flowing back along the catheter, reached the internal iliac artery whose vesical (8) and internal pudendal branches (9) were also opacified. Finally, there was a filling of the urogenital network (10) and of the obturator arteries (11), which serve as collaterals of the deep femoral artery (12) through their anastomoses with the circumflex femoral vessels.

The same situation was observed in Case IV (Fig. 4, *A* and *B*). The occlusion was localized just below the superior mesenteric artery. The renal arteries (2) as well as the superior mesenteric artery (3), were well injected but the branches of the latter were less evident as there already existed a partial occlusion (postmortem examination showed the presence of a free thrombus allowing only the passage of a limited blood flow). Even in this case, the contrast medium reached the left colic artery through Riolan's arch, thus demonstrating a communication with the inferior mesenteric artery.

In all the above mentioned cases, a new blood supply to the distal portion of the colon, which under normal conditions is provided by the inferior mesenteric artery, was established by the collateral pathways.

In Case II (Fig. 2, *A* and *B*) the formation of a collateral system and the restoration of the visceral circulation was more complex and took a longer and less usual course. In this case the aorta was injected at the level of the second lumbar vertebra. The third and fourth lumbar arteries (2) and, through these, the vertebral circle (2″) were well visualized. The lumbar network showed anastomoses with the iliolumbar (3), gluteal (5) and lateral sacral (4) arteries. Filling of the internal pudendal artery (6) was made possible through the latter vessels. A retrograde filling of the

inferior mesenteric artery (8) was obtained through the anastomoses between the lateral sacral artery, the internal pudendal artery, and the hemorrhoidal and vesical network. Apparently, the contrast medium also reached the proximal part of the left colic artery (9). Besides, through anastomoses existing between the lumbar arteries of one side and the corresponding contralaterals (2′), some of the opaque medium filled certain branches of the lumbar arteries of the opposite side, reaching even the pelvic-parietal branches (Fig. 7).

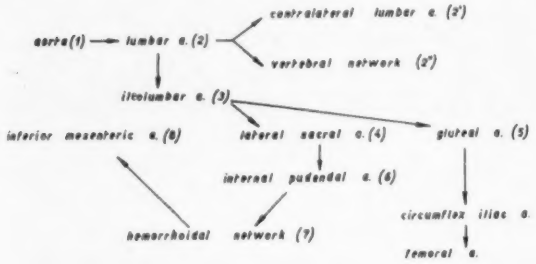

Fig. 7. Another type of collateral circulation to the inferior mesenteric artery through the intermediary of the lumbar network (Case II).

It may be inferred that in this case blood which had been able to flow from the lumbar network to the visceral network of the inferior mesenteric artery could retrogress through the anastomoses between the vessels of the large and small intestine as far as the branches of the superior mesenteric artery. As regards the collateral pathways to the lower extremities, the femoral network was reached through the anastomoses between the gluteal branches (5), the iliac circumflex and the femoral circumflex branches.

In Case V (Fig. 5, *A*, *B* and *C*) a high occlusion of the aorta below the renal arteries was present, owing to a partially free thrombus, which reached the superior mesenteric artery, causing it to be only partly filled (1). One of its right branches, presumably the right colic artery (2), is still recognizable and it can be seen how this vessel is connected with the left colic artery (4) through a long anastomotic arch (3), thus allowing a collateral circulation to the inferior mesenteric artery (5). The terminal branches of the inferior mesenteric artery take part in the formation of the hemorrhoidal network, which is connected on one side with the sacral and on the opposite side with the urovesical network. These connections allow, therefore, an anastomosis with the internal iliac artery (7) at its merging with the external iliac artery (8) and, in some cases, even with the common iliac artery and the femoral artery (9) (Fig. 8).

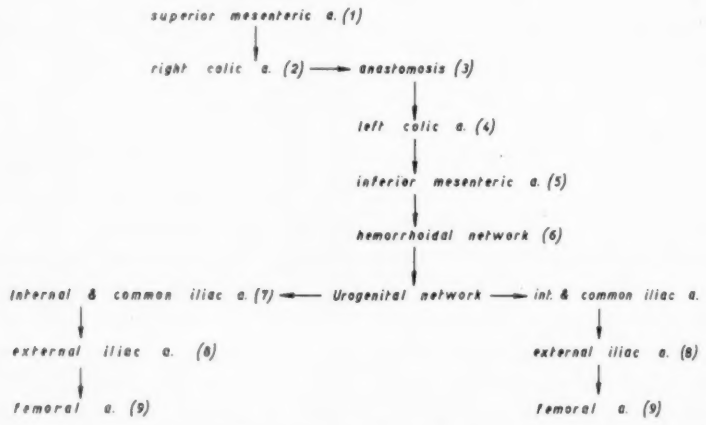

Fig. 8. Type of collateral circulation between the superior mesenteric artery and the femoral artery (Case V).

SUMMARY

Of a total of 104 aortographies through direct puncture of the abdominal aorta, the author has studied 5 cases of thrombosis with complete high occlusion of the abdominal aorta.

The roentgenologic findings are reported, with particular emphasis on the formation of collateral pathways supplying the abdominal viscera as well as the lower extremities.

Via Visconte Modronel
Milano, Italy

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TORTUOSITY AND DEFLECTION OF THE VERTEBRAL ARTERY*

By LEE A. HADLEY, M.D.
SYRACUSE, NEW YORK

EXCESSIVE tortuosity or buckling of the carotid, subclavian and the innominate arteries is described by various writers,[1,10,11] but tortuosity of the vertebral artery resulting in destructive bone changes seems to have remained unrecognized. The condition is clinically significant, however. Such deviations may be congenital or may result from arteriosclerosis or luetic disease. Kovács[13] has published the angiogram of a patient with such a vertebral artery but without bone erosion. Kelly[11] collected 85 cases with prominent, pulsating internal carotid arteries, 21 of these in patients under ten years of age. These latter examples he considered to be congenital in origin. He described no cases of tortuous vertebral artery.

MATERIAL

In a group of 21 cadavers aged forty-five to ninety-one years, 4 examples of tortuosity of the vertebral arteries with bone destruction were encountered. The vessels shown in Figures 1, 2 and 3 were arteriosclerotic and roentgenograms revealed various degrees of bone destruction. Cadaver number 4, the youngest, was a victim of cerebrospinal syphilis. In each instance, sections of the vertebral artery showed degenerative changes in the vessel wall. There has been no opportunity for the anatomic correlation of similar roentgenographic findings in living patients. However, roentgenograms (Fig. 6 and 7) of patients made in the oblique projection demonstrate such bone changes. A pedicle may be entirely lacking, or the limit of the area of destroyed bone may be indicated by a fine curvilinear shadow. Two of the 3 instances which I reported in 1946 as congenital absence of a cervical pedicle were

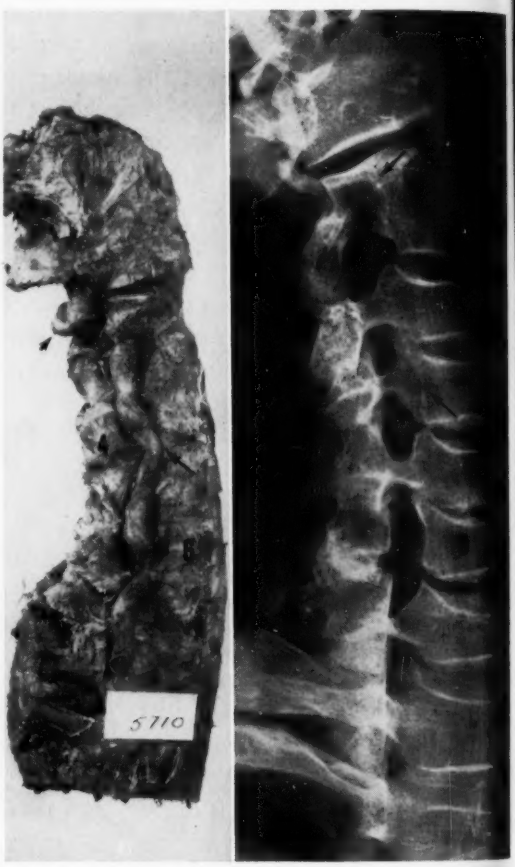

FIG. 1. Male, aged ninety-one. Sclerosis of peripheral vessels, Wassermann test negative. Mitral disease. Blood pressure 149/78. Section of vertebral artery showed thinning of both the media and the intima. A pulsating tortuosity of the right vertebral artery has completely destroyed the sixth cervical pedicle. Curvilinear bone erosions from arterial pressure are also visualized on the posterior surface of the C_4 body and within the second transverse process (arrows). The latter was still incompletely uncovered when the photograph was made. The fourth posterior articulation displaces the artery forward (4). One may note a complete loop of the vertebral artery behind the atlanto-axial joint (small arrow head).

* These studies were assisted by the Departments of Anatomy and Photography, New York State School of Medicine, Syracuse, New York.

Fig. 2. Female, aged eighty-three. Wassermann test negative. (*R*) Right side. Pressure destruction of the posterolateral surface and a part of the pedicle of C6 and the lateral mass of C2 (black arrows). The opposite oblique projection (O) of the right side shows the cortices of the normal pedicles en face as round shadows (P). Erosion of the anterior surface of the sixth pedicle, however, is indicated by a defect of the corresponding round shadow (white arrow).

Fig. 2. (*L*) Left side. Tortuosities at the C2, C4 and C6 levels (arrows).

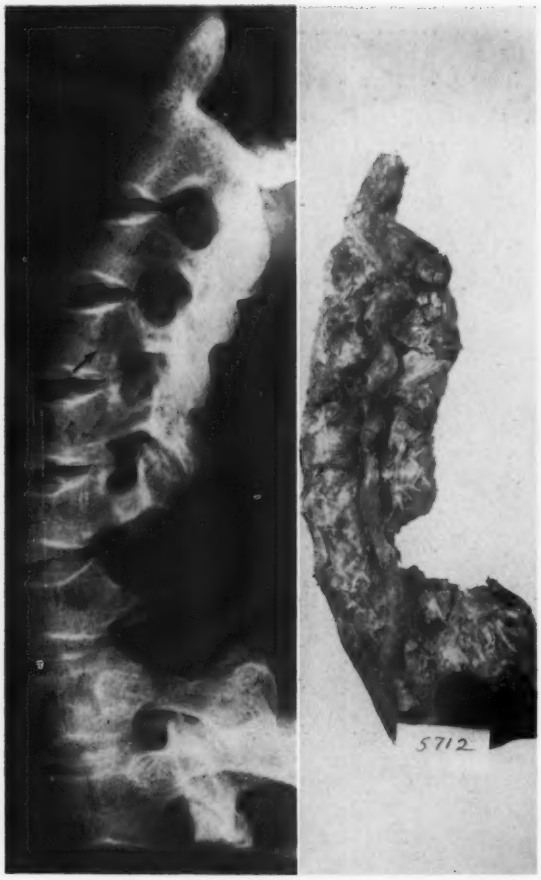

Fig. 3. Female, aged sixty-seven, unmarried. No Wassermann report. Blood pressure 170/10 Tortuosity of the left vetebral artery eroding the fourth cervical body (arrow). Since a limiting cortical reaction about the erosion is absent in this case, angiography would have been necessary to visualize the bone deficiency. The section showed marked degenerative thinning and calcific deposit within the media.

Fig. 4. Male, aged forty-five. Cerebrospinal syphilis. Infection ten years before death. Blood pressure 160/96. (*R*) Right side. Vertebral artery follows a normal course except for the tortuosity within the lateral mass of C2 (arrow). A section taken at this point showed marked thinning of the vessel wall; the intima was absent on the outside of the curve but no luetic changes were seen. Note the normally close relationship of the cervical spinal roots to the posterior surface of the artery.

Fig. 4. (*L*) Left side. Tortuosity eroding C3 (arrow). The arterial curvature within the left lateral mass of C2 is about normal as contrasted with the tortuosity within C2 on the right side.

Fig. 5. Female, aged seventy-two. Arteriosclerosis. Wassermann test negative. Left half of cervical spine. Angiogram of vertebral artery. (*1*) The posteroanterior projection shows a covertebral spur below C_3 producing lateral deflection of the artery (arrow). (*2*) The lateral roentgenogram made at full limit of dorsal extension shows the third posterior joint also deflecting the artery forward (arrow) as described by Kovács.[13] This did not occur with the specimen in the neutral or anteflexed positions and corroborates the observations of Lewis and Coburn.[14] These authors report 11 patients showing angiographic evidence of interference with vertebral artery circulation and complaining of various types of head pain, dizziness, nausea and eye symptoms. The symptoms occurred only with the neck in dorsal extension and are held by various observers to be caused by circulatory and/or sympathetic disturbance.[4,9,18]

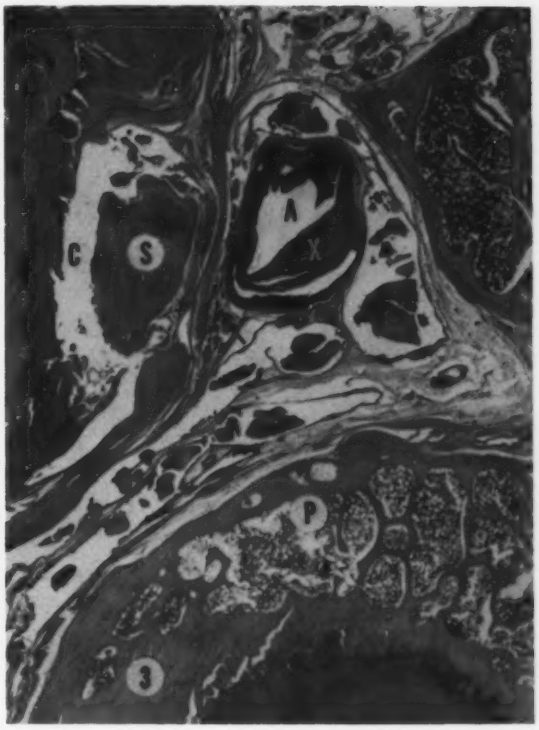

Fig. 5. (*3*) The transverse section, ×8, is taken through the artery, covertebral spur and posterior articulation at the plane indicated by the lines on the roentgenograms in Figure 5, *1* and *2*. C is the covertebral joint space between C_3 and C_4. The rectangular shaped artery (A) is grossly distorted by the covertebral spur (S) and the posterior articulation (P). The vertebral artery sections from other cadaver specimens were usually round or slightly oval. This one contains an organized atheromatous plaque (X) partially separated from the wall of the vessel, a condition described by Hutchinson and Yates.[9] The arrow indicates the site of three sectioned sympathetic nerves. Even in the presence of deflecting pressure as observed in this and other specimens, the vertebral sympathetic continues to follow the artery, usually on its posterior surface. Hoffman and Kuntz[8] state that the vertebral nerve with its inconstant ganglia may extend to the first or the second cervical root and then the vertebral sympathetic plexus becomes continuous with the intracranial basilar plexus above. The vertebral artery is accompanied by a rich network of veins.

probably examples of vascular tortuosity. Steinbach and associates[16] have published a roentgenogram of the cervical spine of a twenty-eight year old woman in whom the right seventh pedicle was absent. The vertebral arteriograms, however, revealed a normal appearing vessel and no pathology was found at operation. The condition was probably congenital absence of the pedicle. Hodges and Holt[7] report a somewhat similar experience. The importance of these findings lies in the possibility that they may be mistaken for the results of tumor pressure or even trauma.

DISCUSSION

Various authors[3,12,14,19] have described vertebral artery deflection and the symptoms resulting from pressure upon this ves-

Fig. 6. Male, aged thirty-five. Blood pressure 118/70. Originally diagnosed elsewhere as a fracture. This is a case which I reported[5] as congenital absence of the fifth left cervical pedicle. The clearly seen left fifth transverse process (white arrow) normally arises from the pedicle early during gestation. In the light of the recent anatomic findings, however, I now believe this to have been really an example of tortuosity of the left vertebral artery with destruction of the fifth pedicle, similar to the condition seen in Figure 1. The patient had bilateral involvement. A restudy of the right side now reveals, for the first time, an apparent somewhat less extensive tortuosity with bone destruction involving the right sixth vertebral body (black arrow). Compare this with Figure 2*L*.

sel. Bony spurs from hyperplastic covertebral joints or from grossly arthrotic posterior vertebral articulations may deflect and compress the artery and its accompanying nerve and plexus. This deflection and the resulting symptoms are exaggerated by rotation and/or dorsal extension of the neck. In Kovács's[13] later monograph this condition is shown beautifully by laminagrams.

Various examples of pressure deflection were also encountered in the cadavers studied. For instance, in cadaver 5 the left vertebral artery was deflected laterally by a covertebral spur at C3 (Fig. 5, *1* and *2*). An arthrotic posterior articulation also deflected the artery forward at this same level upon dorsal extension but not when the cervical spine was flexed anteriorly. Transverse section of the artery at this point (Fig. 5, *3*) showed partial occlusion by an organized atheroma. Another example was reported in a previous publication[6] as a deflection caused by a covertebral joint.

The vertebral artery is an important link in the collateral cerebral blood supply. Angiography is necessary to visualize pressure deflection or atheromata of this vessel.[2,14,15,17] The symptoms of these changes are said to result first, from interference with the important circulation to the hind brain, and, secondly, from pressure upon the sympathetic nerve and the plexus which surrounds this vessel.[4,9,14,18]

The vertebral artery arises from the subclavian. From the sixth vertebra upward it

Fig. 7. Female, aged thirty-one. Blood pressure 130/70. This patient had complete loss of the left sixth cervical pedicle with displacement of the adjacent side of the neural arch backward and downward. The lateral portion of the transverse process external to the foramen of the vertebral artery has remained intact (arrow). The condition probably developed slowly and may have been congenital. (Courtesy, Dr. Martin S. Abel, San Francisco, California.)

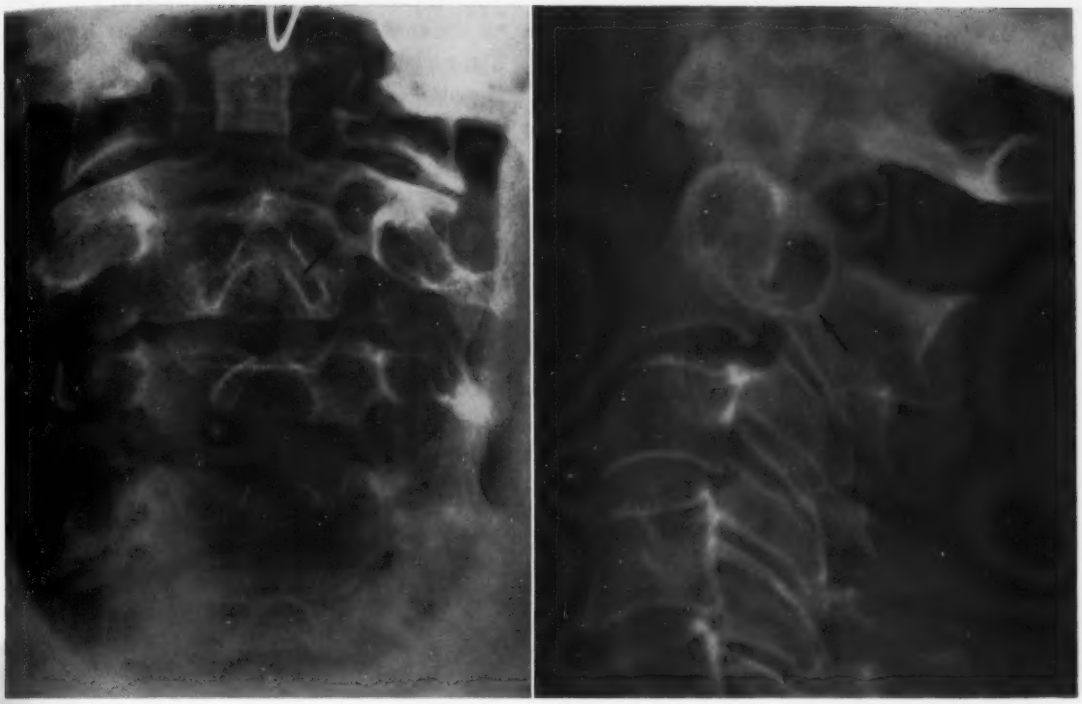

Fig. 8. Female, aged fifty-six. Blood pressure 185/85. A curvilinear erosion (arrows) is seen in the anteroposterior and lateral projections of the second cervical vertebra adjacent to the location of the vertebral artery. The appearance is somewhat similar to that in Figure 4*R*. The C2 left lateral mass and pedicle have been partially eroded, probably by a tortuosity of that vessel. (Courtesy, Dr. Samuel Gingold, Syracuse, New York.)

is a captive vessel, coursing through its foramina within the transverse processes. In the lateral roentgenogram the neck seems much longer upon forward flexion than with dorsal extension. However, since the artery lies in the central axis of movement between adjacent vertebrae, the distance from the inferior surface of the sixth to the upper surface of the second cervical transverse process measures the same in both positions. On the other hand, extremes of rotation and flexion occur at the upper cervical region. Three normal curves in the vessel help to compensate for movements at this level. The lowermost curve is backward and laterally between the third and second transverse processes. Next, the artery curves forward within the second transverse process, and, finally, on the upper surface of the first neural arch, it encircles the posterior margin of the articular mass. As a result of arterial lengthening any of these curves may become exaggerated.

SUMMARY

Tortuosity of the vertebral artery may result in demonstrable bone erosions visualized on the roentgenogram of the cervical spine made in the oblique projection. Three of the patients herein mentioned were less than thirty-six years of age. The condition may be a congenital one or may result from later arterial changes.

Also, the vessels may be deflected and distorted by extrinsic pressure of bony spurs at the covertebral or apophyseal joints. The decreased blood flow and/or pressure upon the sympathetic plexus may be a factor in producing neurologic symptoms.

925 State Tower Building
Syracuse, New York

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SPLENOPORTOGRAPHY: SOME ADVANTAGES AND DISADVANTAGES*

By MELVIN M. FIGLEY, M.D.
ANN ARBOR, MICHIGAN

NEARLY six years have elapsed since Abeatici and Campi[1] described the possibility of roentgenographic visualization of the portal vein and its branches by intrasplenic injection of contrast material. In this interval percutaneous splenoportography has been widely practiced and sufficient experience has been recorded for an adequate assessment of its advantages and disadvantages. This report, based upon personal experience with 125 attempts at percutaneous splenoportography and a review of several large series of cases,[7,23,31] renders an accounting from the points of view of technique, consistency of results, hazards involved and interpretation of certain observations. Thoughtful appraisals of the clinical usefulness of the method have been presented elsewhere.[12,15,17]

TECHNIQUE

An important advantage of the procedure as it is commonly conducted is its simplicity in performance. Preparation of the patient usually consists of omission of the previous meal and sedative premedication. Immediate surgical intervention following the procedure, though carried out in some centers,[10,16,18,29] is not regularly considered necessary on the basis of the risks involved. Local anesthesia is regarded as sufficient except in children. Splenic enlargement facilitates the examination but successful punctures of normal spleens have been made without elaborate localizing procedures. Injection can be made rapidly enough by hand and need not be accurately timed.[4] It requires only a short needle, large[18] or small,[31] plastic adapter tubing and syringe. The contrast material may vary somewhat as regards dose and concentration. Ordinary roentgenographic equipment and manual cassette changing have been adequate, but those with the greatest experience advocate the use of some automatic serial filming device.[7,12,23,31] Although this requires that the procedure be performed in the radiologic department, it provides a more elaborate film record and the opportunity for preoperative planning based on the results.

The technique at the University of Michigan[19] may be regarded as "controlled," in contradistinction to the "blind" puncture and injection during apnea.[4] A preliminary roentgenogram is obtained to check the positioning and exposure factors and to localize the spleen in frontal projection in relation to the anticipated puncture site, usually in the ninth interspace in the mid or posterior axillary line. A short No. 18 gauge needle is slowly introduced into the spleen, apnea being required only during its manipulations. The flow of venous blood and wide oscillations of the needle butt suggest that it is properly located in the spleen. Frequently it is advanced another 1–2 cm. to be certain it is well within the pulp. Roentgenography after a 5 cc. injection is sometimes done if there is difficulty or uncertainty. The definitive dose of 25 cc. of 70 per cent urokon is injected manually in five to eight seconds and serial exposures are made at 1.3 films/second for twenty seconds (for visualization of the hepatic veins exposures as late as thirty-five seconds may be necessary[4]). These maneuvers prolong only slightly the duration of the procedure and, as will be seen in the following sections, provide no additional hazard.

CONSISTENCY OF RESULTS

One of the disadvantages of the procedure is the unpredictability of success

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Table I

SPLENOPORTOGRAPHY—CAUSES OF FAILURE

Failure to find the spleen—no injection	4
Failure of transport of contrast material or extrasplenic injection	11
Roentgenographic failure	3
Unknown	3

with the method as described. In 125 attempts on 111 patients* we must acknowledge 21 (16 per cent) outright failures. Some of these can be ascribed to inexperience and the fact that no test injection was used but some have occurred even recently in experienced hands. The causes of failure are listed in Table I.

Failure to find the spleen occurred only in those patients without palpable spleens, a feature encountered in about 40 per cent of the patients, many of whom had successful studies. Failure of transport and extrasplenic injection may be difficult to distinguish unless the bowel, stomach, pleura or kidney are recognized (injection into these has not proved harmful). Failure of transport is probably the less frequent, but it did occur in a patient who at laparotomy, performed immediately afterward, showed evidence of having had a proper puncture. It may have occurred in another patient with splenic artery occlusion caused by pancreatic neoplasm but recent experimental work by Bergstrand and Ekman[3] casts doubt on this assumption. Extrasplenic injection was observed principally in those patients without palpable spleens. Probably it can be completely avoided only by test injections. Organs, other than the spleen, may drip venous blood and move the needle butt with respirations simulating splenic puncture.

Other authors with experience in comparable or larger series of cases report approximately the same rate of failure (Table II). Bergstrand and Ekman[4] described in detail 20 extrasplenic injections, 17 of 137 "blind" punctures and 3 of 35 "controlled" punctures, more often in those cases without splenomegaly. Gvozdanović and Hauptmann[23] noted 6 extrasplenic injections in 61 studies in patients, all with splenomegaly. Steiner, Sherlock and Turner[31] report 13 failures in 100 "controlled" injections in patients with splenomegaly.

Another disadvantage is the significant number of suboptimal examinations, some 22 of our attempts (16 per cent) resulting in only fair or poor roentgenograms, although they were adequate for certain purposes. Part of these difficulties were due to exceptionally large patients (3) and avoidable roentgenographic error (5) but the bulk were due to partial loss of the contrast medium by extravasation from the spleen (14). This may be a fault of the "controlled" injection technique since during the respiratory motion the needle may become less tightly gripped by the spleen. Bergstrand and Ekman[4] substantiate this and report their observation of decreasing pressure as the needle lies in the spleen. Furthermore, their experience with the two methods, 7 per cent (10 of 137) extravasations with "blind" injection and 27 per cent (13 of 35) with "controlled" injection favors the former.[4] Gvozdanović and Hauptmann[23] had a somewhat higher rate of extravasation, 16 per cent (10 of 61), with the "blind" technique. Aside from its negative value roentgenographically, extravasation is undesirable as it is decidedly painful whereas a completely intrasplenic injection is not. We have tried unsuccessfully to eliminate it by using a needle with multiple perforations at the tip intended to "spray"

Table II

SPLENOPORTOGRAPHY—FAILURES

			Per Cent
Gvozdanović and Hauptmann[23]	1955	6 of 61	10
Steiner, Sherlock and Turner[31]	1957	13 of 100	13
Bergstrand and Ekman[4]	1957	21 of 175	12
University of Michigan	1957	21 of 125	16

* Many of these patients as well as others from another hospital were considered in an earlier report.[12]

the material into the spleen. There is little evidence that extravasation is related to needle size or speed of injection.

HAZARDS INVOLVED

Fortunately, the single major hazard of splenoportography, namely serious postpuncture bleeding, is rather infrequent. Doubtless, there is some bleeding in many cases as evidenced by the fact that explorations immediately following the procedure have disclosed 50–200 cc. of free blood. Two of our patients were still bleeding at laparotomy thirty to sixty minutes later and another had accumulated 300–400 cc. of blood near the spleen. However, to date none of our patients has had laparotomy because of bleeding, although one patient with unsuspected hypersplenism and thrombocytopenia required a transfusion of 1,500 cc. of blood for control of hemorrhage.

Other investigators have had about the same experience with postpuncture bleeding. Gvozdanović and Hauptmann[23] report 2 instances of bleeding in 61 studies, 1 requiring transfusion. Two of the patients of Steiner, Sherlock and Turner[31] required transfusions, one at twenty-four hours and one at five days after puncture. At least 6 patients have required[9,15,20,28,32] splenectomy for control of hemorrhage due in each case to splenic tear. One patient with granulocytic leukemia died of splenic rupture complicated by esophageal and gastric hemorrhage.[30]

In 4 of Bergstrand and Ekman's[4] 175 cases, splenoportography was followed by shock, suggestive of intra-abdominal bleeding. Each was treated successfully by transfusions. At subsequent laparotomy in 2 of these at four and ten days, there was no evidence of hemorrhage.

No complications have been reported from injection of the contrast medium into other organs such as the stomach, colon or pleural cavity or from extravasation aside from pain. Furthermore, no deterioration of liver function has been ascribed to an uncomplicated procedure.

INTERPRETATION

The interpretation of splenoportograms seems relatively straightforward but certain pitfalls and limitations have been recognized. Two general types of visualization are obtained. One type shows only the splenic vein fed by one or more hilar tributaries and the portal vein and its branches (Fig. 1*A*). The insertions of other tributaries may be visible but there is no sustained flow in them. Such roentgen images we regard as indicating no portal obstruction or portal hypertension although this does not exclude the possibility of disseminated hepatic disease.

The other type shows sustained hepatofugal flow in portal and splenic tributaries (Fig. 11). These images we consider as suggestive of some portal (or splenic) obstruction. While hypertension is generally present also, the dilatation of such tributaries —collateral circulation—may be so great as to compensate for the obstruction and render the system normotensive. A few such cases have been described.*[13,31]

It is important to determine the influence of injection technique upon portal hemodynamics if these concepts are to be accepted. Dreyer[14] first studied this experimentally in 6 dogs and found no measurable increase in pressure by intrasplenic injection of 20 cc. of saline or contrast medium. More recently, Bergstrand[7] has found the same except that thirty to sixty seconds later there may be slight elevation of pressure—a time when the contrast material will have already cleared from the larger veins. He showed that variations in the rate of injection had no influence on the velocity of flow, vein width or appearance of collaterals. The experimental evidence then suggests that the splenic pulp effectively cushions the portal system from

* Since this paper was prepared, a report has appeared correlating intrasplenic pressure and the visualization of portal tributaries. While the concepts expressed above are generally true, exceptions do occur. Portal hypertension may be present without visible collateral circulation. Collateral circulation may be visible without measurable portal hypertension. Turner, M. D., Sherlock, S., and Steiner, R. E. Splenic venography and intrasplenic pressure measurement in clinical investigation of portal venous system. *Am. J. Med.*, 1957, *23*, 846–859.

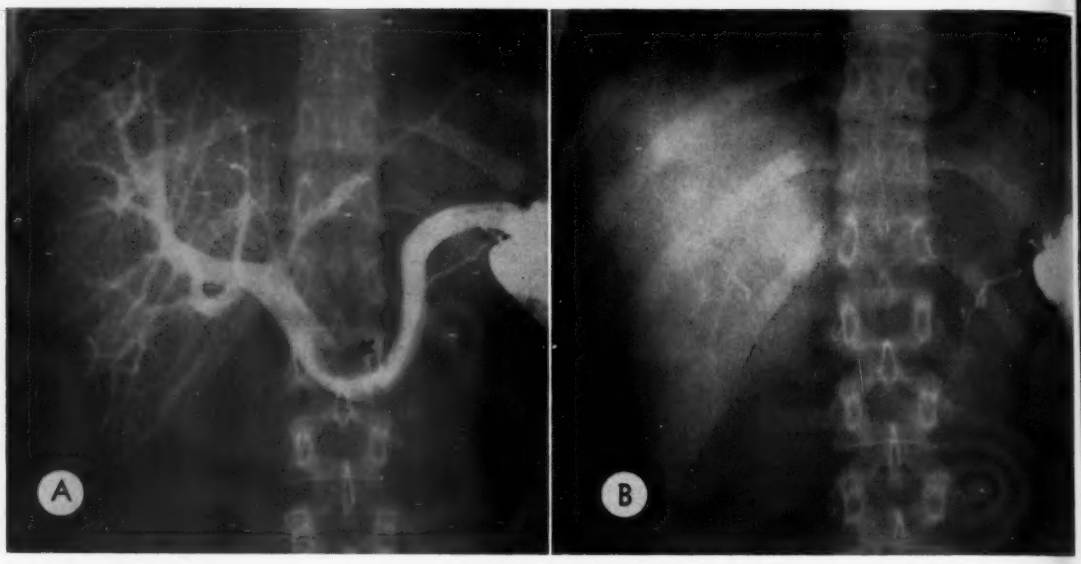

Fig. 1. Normal splenoportogram. Female, aged thirty, with subacute hepatitis. (*A*) Splenic, portal and intrahepatic veins. Typical mixing defect in portal vein (arrow) where nonopaque blood from superior mesenteric vein enters. No flow-away from liver in portal or splenic tributaries. (*B*) Hepatogram of uniform density.

changes in pressure or flow during splenic injection.

Similar observations in man are lacking but the occasional fortuitous direct puncture of the splenic vein provides useful information (Fig. 2, *A* and *B*). Under this

Fig. 2. Direct injection into the splenic vein: (*A*) at onset of injection; (*B*) four seconds later. Intense opacification of segment of spleen. The flow into inferior mesenteric and coronary veins (arrows) progressed no farther than shown here.

condition maximum change in dynamics might be expected as the interposition of the splenic pulp is eliminated. In such cases we have seen brief unsustained hepatofugal flow into tributaries and interpret this to mean brief elevation of pressure during injection but not thereafter. Serial roentgenograms are of course necessary for such observations. Bergstrand and Ekman[3] support this by reporting that direct splenic vein injection in dogs elevates the pressure 1–3 cm. of saline during injection and fills tributaries that were not previously filled by splenic injection.

Clinical experiences also support the concept that reversed flow in tributaries means portal (or splenic) obstruction. As yet no one has reported this as a normal occurrence. Bergstrand and Ekman[5] found it only in cases with other evidence of portal hypertension, clinical or manometric. In our pathologically proved material we have never seen it in the absence of some abnormality.[12] Of course, this is not to say that a normal pattern excludes significant disease. Of 42 cases of liver cirrhosis in which splenoportograms allowed reliable assessment of filling of tributaries, retrograde flow (portal hypertension) was lacking in 7 (Fig. 3). It has also been absent in some cases of massive involvement of the liver by neoplasm.

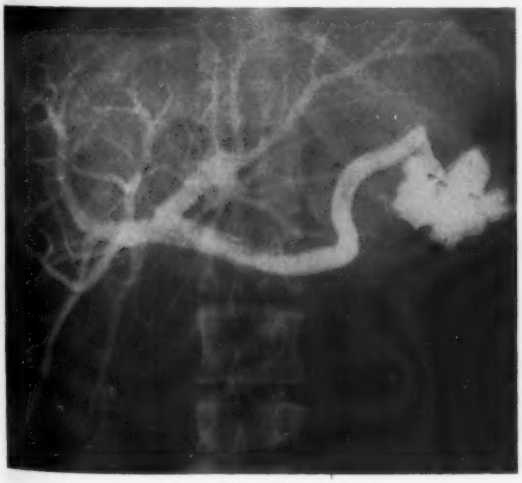

Fig. 3. Portal cirrhosis of the liver. Female, aged thirty-seven. Liver is enlarged but there is no hepatofugal flow to indicate portal obstruction.

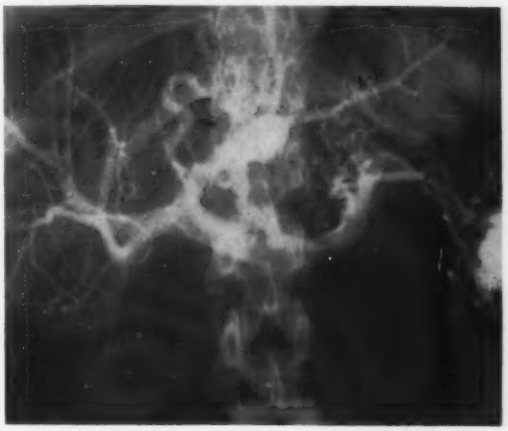

Fig. 4. Suprahepatic portal obstruction due to sarcoma of the right atrium occluding the inferior vena cava and hepatic veins. Female, aged thirty-two. Retrograde flow into short gastric and paraumbilical veins (arrow) indicates portal hypertension.

Accepting the concept that reversed flow in tributaries (collateral circulation) means portal obstruction and probable hypertension, it is necessary to recognize supra, intra and extra (infra) hepatic locations of such obstruction. Any impediment to outflow from the hepatic veins might be regarded as suprahepatic obstruction and may be expected to produce hepatic enlargement and hypertension (Fig. 4). By splenoportography alone this may be difficult to distinguish from intrahepatic disease unless there is some direct evidence of the latter. Cirrhosis and diffuse infiltrative processes in the liver, such as Gaucher's disease, may present the same appearance of large liver with portal hypertension. But more often the cirrhotic liver is smaller than normal and typically deformed portal venous branches may be visualized. Attenuation and tortuosity of these have been shown by corrosion preparations[26,27] and are seen in the hepatograms of about two-thirds of cases.[7,31] These represent the specific splenoportographic sign of cirrhosis (Fig. 5).

The site of obstruction, whether intra or extrahepatic, is of great concern to the surgeon as regards his choice of portacaval or splenorenal shunting for the control of

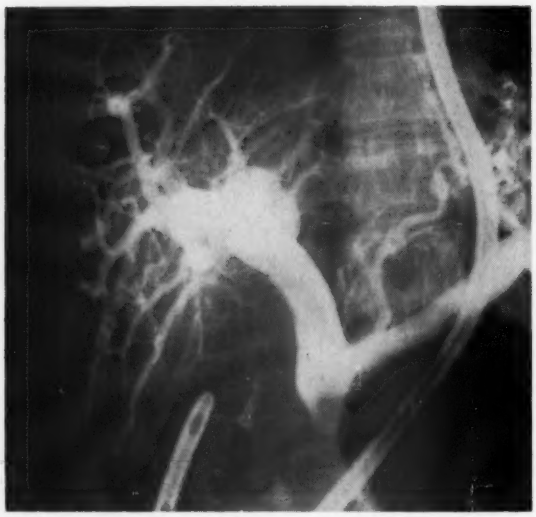

Fig. 5. Cirrhosis of the liver. Female, aged sixty-two, with active esophageal hemorrhage. Sengstaken-Blakemore tube in place, gastric balloon inflated, esophageal balloon deflated. Note the large caliber portal vein and crowded tortuous attenuated intrahepatic branches, typical of shrunken cirrhotic liver. Portal obstruction is indicated by retrograde flow into short gastric and coronary veins.

bleeding varices. In fact this determination is one of the principal indications for the splenoportography. It has been pointed out, however, in both experimental work[3] and by clinical experience,[3,16,25,31] that the contrast material may be diverted completely into the collateral circulation, thereby not reaching the portal block. Thus, a patent portal vein may not be visualized. But certainly the occurrence is not sufficiently frequent to detract from the general usefulness of the procedure. In 46 patients with liver cirrhosis, examined adequately as regards portal vein patency, we found the vein patent in all but one case. Although portal vein thrombosis occasionally complicates cirrhosis, we have not encountered it. In this regard serial roentgenography has been of definite help in distinguishing mixing or "streamline" defects from thrombi. Furthermore in 9 cases diagnosed as extrahepatic obstruction due to portal vein thrombosis, liver biopsy was normal in each case, excluding diffuse parenchymal disease as the cause of the obstruction. Thus only one "false positive" extrahepatic portal occlusion has been encountered (Fig. 6). When this is suspected, it may be verified by operative portography as advocated by Du Boulay and Green,[15] and by Rousselot *et al.*[29]

The second principal use of splenoportography has been for the purpose of detecting expansive processes in the liver, the signs being occlusion and deformity of the portal branches and defects in the hepatogram—that phase of uniform liver density produced by sinusoidal filling (Fig. 1*B*). Of these, the former signs are of greater value since a sufficiently dense hepatogram is not regularly obtained. We have obtained useful information in 16 patients with liver disease other than cirrhosis with pathologic verification in 13. Two large cysts and 8 sizable tumors (5 proved) were recognized, but disseminated disease with small local lesions (neoplasm, 2; histoplasmosis, 1; and Gaucher's disease, 1) caused no alteration

Fig. 6. Postnecrotic cirrhosis and hepatoma of caudate lobe. Male, aged seventy-two. The tumor in the caudate lobe compressed the portal vein which was otherwise patent and free of thrombus. Splenoportogram shows total diversion of contrast material into a huge coronary vein (arrow). The patent portal vein is not filled—but in this instance due partly or perhaps wholly to compression by tumor.

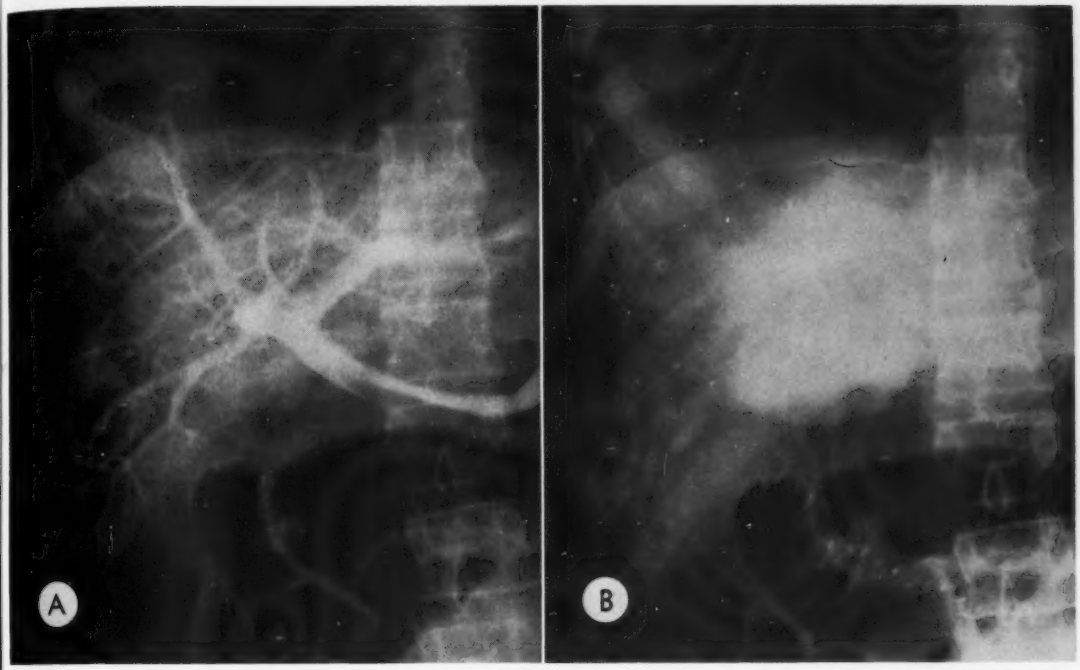

Fig. 7. Liver rupture. Male, aged twenty-two. Eight weeks after injury. (*A*) Most of the intrahepatic branches seem to be intact. (*B*) The hepatogram is strikingly nonuniform with intense opacification of the area of the caudate lobe. This degree of nonuniformity has not been seen previously except in the presence of neoplasm.

except hepatic enlargement and, in the instance of Gaucher's disease, portal hypertension. Others have mentioned the same difficulty in locating disseminated neoplasm when the nodules were small.[2,6,22] One case of hepatic tumor (Fig. 6) and another of ruptured liver (Fig. 7, *A* and *B*) had unique features.

While venous occlusions and their corresponding hepatographic defects are usually due to neoplasm, alterations in the circulation following liver rupture resemble them (Fig. 7, *A* and *B*). Furthermore, total filling of the portal branches does not always occur in the normal, as the left lobe often fills later and less intensely than the right (Fig. 8, *A* and *B*). This can be attributed to the high specific gravity of the contrast material directing it to the more dependent right lobe as the patient lies in a supine position. It can be overcome by the use of the prone position.[3]

As far as defects in the hepatogram are concerned, some qualification of their meaning must also be made. On occasion, in an apparently normal liver, opacification is not uniform (Fig. 8*B*). Defects have been observed where there was no palpable abnormality (Fig. 9). Thus, the splenoportographic signs of hepatic neoplasm must be regarded in the light of the clinical situation. They are presumptive but not infallible evidence.

The third major use of splenoportography has been in search of evidence of pancreatic neoplasm which may displace or invade the portal and splenic veins. The course of the latter is so variable that slight alterations are not likely to be recognized although local compressive or invasive lesions can be seen and may result in some splenic hypertension (Fig. 10). We have studied a number of patients in whom pancreatic neoplasm was considered and have collected 12 cases with pathologically verified pancreatic or peripancreatic disease: 4 with primary carcinomas, 5 with regional neoplasm involving the pancreas and 3 with pancreatic cysts. In only 4 of the 9 cases of neoplasm were there convincing signs of

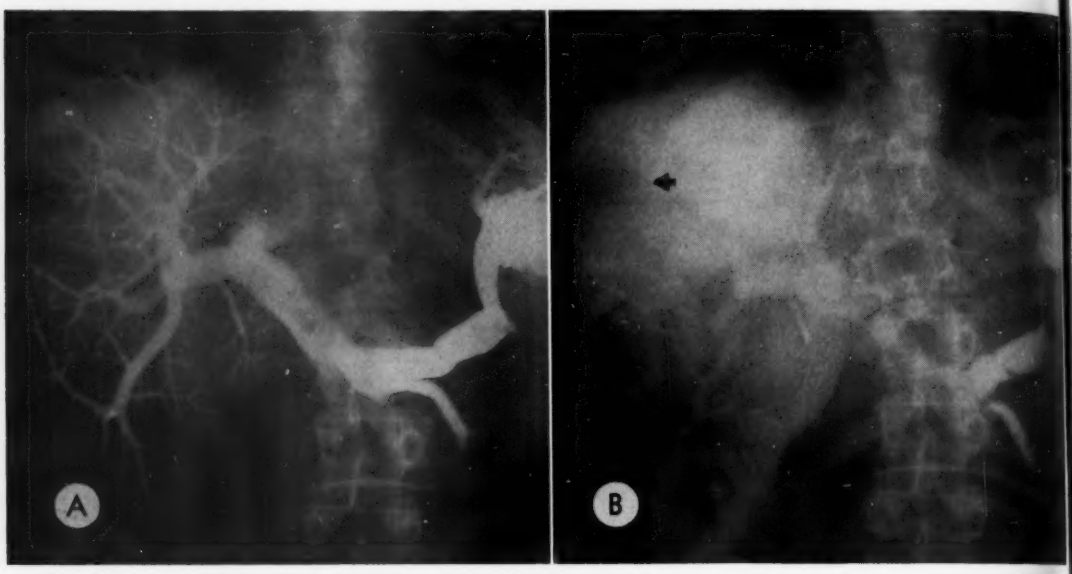

Fig. 8. Common bile duct stricture with early biliary cirrhosis. Female, aged forty-nine. (*A*) Normal appearing branches in the right lobe. No filling of left lobe (not grossly abnormal at surgery). (*B*) Hepatogram is nonuniform (arrow). Flow into the inferior mesenteric vein is unsustained.

tumor (Fig. 10 and 11), but in the cases with cyst some splenic vein obstruction was noted, twice due to the mass and once due to splenic vein thrombosis apparently related to accompanying pancreatitis. In these cases, cysts in the head of the pan-

Fig. 9. Metastatic carcinoma of the liver. Male, aged forty-six. Right colectomy for adenocarcinoma five years previously. At exploration the left lobe of the liver was replaced by neoplasm, but there was no palpable abnormality in the region of defects in the tip of the right lobe and caudate lobe (arrow). At autopsy seventeen months later only one huge metastasis was found in the center of the liver. The origin of these defects is unknown.

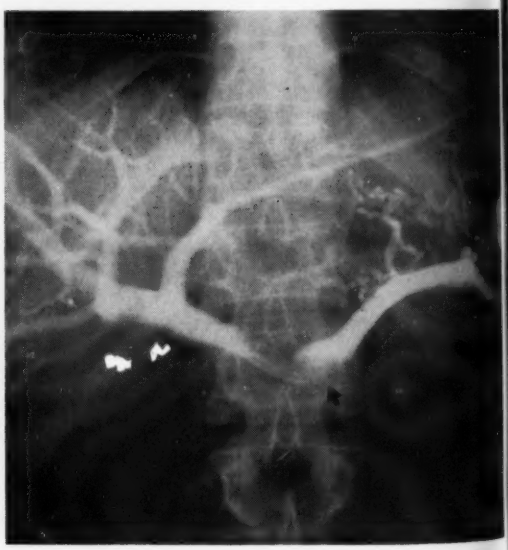

Fig. 10. Postoperative recurrence of carcinoma of ampulla of Vater. Female, aged thirty-nine. Splenic vein compression (arrow) with some splenic hypertension as shown by filling of short gastric veins. The splenic hypertension indicates that this is not a simple mixing defect. No definite indication of venous invasion—a "benign" appearing lesion.

creas were not detected. Thus it would appear that sizable pancreatic neoplasms may not be recognized, particularly in the head —although there is no denying evidence of invasion—and further it appears that benign processes may produce the same nonspecific signs of compression and thrombosis that occur with malignancy.

DISCUSSION

The literature concerning percutaneous splenoportography is already voluminous, much of it containing enthusiastic reports of its clinical usefulness. Only a few factually support opinions regarding details of technique, hazard and interpretation. Most of these have been collected in the foregoing.

These show that successful examination is thwarted in 10–16 per cent of attempts due to failure to puncture the spleen or inject into it properly, at times even when it is enlarged. Thus the present techniques, while desirably simple, need modification in order to insure a more exact prepuncture estimation of the splenic position, size and thickness, if this failure rate is to be lessened. This is particularly true if the spleen is not enlarged. In such patients the "controlled" puncture technique supplemented by test injections is probably preferable to the "blind" technique.

When the spleen is successfully punctured, injection should be made as promptly as possible in order to reduce backward flow along the needle, such extravasation being the principal cause of suboptimal opacification. This occurs in about 15 per cent of attempts and is relatively uncontrollable as well as painful. It would be distinctly helpful if the splenic vein or its tributaries at the hilus could be consistently punctured for injection, since as of the moment a painful examination is likely to be substandard or unsuccessful.

Splenoportography seems safe enough considering the fact that splenic hemorrhage occurs in only about 2 per cent of patients and the diagnostic usefulness of the procedure is greatest in such serious

FIG. 11. Carcinoma of the pancreas. Male, aged fifty-four. Neoplastic invasion and near total occlusion of the portal vein (arrow). Extensive collateral circulation, especially in the coronary and esophageal veins.

situations as the determination of the cause of gastrointestinal hemorrhage, the origin of portal hypertension, and the presence of hepatic and pancreatic neoplasms. Major surgical intervention frequently hinges upon the information it provides. It seems immaterial whether the "blind" or "controlled" technique is employed as regards the risk of bleeding but, understandably, bleeding occurs more often from enlarged hypertensive spleens. While in proved cases the cause has been a splenic tear or rupture rather than a coagulation deficit, our experience suggests the wisdom of correcting thrombocytopenia and hypoprothrombinemia as much as possible beforehand. Furthermore, this deficit is not very important, for the spleen can be sacrificed to control hemorrhage if necessary. Technical errors do not lead to irreparable damage of vital organs as in some other angiographic procedures, such as translumbar aortography.

In the matter of interpretation there is incontrovertible evidence that portal hemodynamics are not altered by splenic injection of contrast material. Thus, the ob-

servation of sustained reverse flow in tributaries positively identifies some type of obstruction. This has allowed some broadening of our concept of portal hypertension for it has been shown to accompany many lesions, supra, intra and extrahepatic in location. While the principal clinical expression of this hypertension—hemorrhage—is almost confined to cirrhosis and extrahepatic portal thrombosis, hemorrhage occurring with other lesions, such as carcinoma of the pancreas,[24] may be explained on the basis of portal obstruction and hypertension (Fig. 10).

Collected experiences suggest that the most rewarding application of splenoportography is the study of portal hypertension in its narrower sense, especially for the differentiation of intra and extrahepatic block relative to its surgical treatment. The uncommon pitfall of total diversion of the contrast material from a patent portal vein does not invalidate this indication. Other occasionally valuable applications include the positive demonstration of portal hypertension in cases of unexplained gastrointestinal hemorrhage, evaluation of patency of portacaval shunts and the recognition of cirrhosis. Experiences with these applications relative to the results of conventional roentgenologic examination, occluded hepatic vein catheterization and other clinical procedures are fully described elsewhere.[17,31]

As regards the detection of hepatic and pancreatic neoplasms, the method has not been consistently successful. The inconstancy of recognizable alterations in the veins by these diseases and the inconstancy of complete opacification of the normal hepatic branches are partial explanations. Correction of the latter may be effected by examination in the prone position to insure the best possible filling of the left lobe. Furthermore, since a good hepatogram is essential, it may be advisable to use a somewhat larger amount of contrast material, perhaps 40–50 cc. Other methods, of course, can be employed in these conditions—liver biopsy, transhepatic portal venography,[8] retrograde hepatic venography,[11] scanography,[21] etc., but discussion of their merits as compared to those of splenoportography is not within the scope of this paper.

CONCLUSIONS

A critical study of 125 attempts at percutaneous splenoportography and review of several thorough analyses by others, together totalling nearly 500 such examinations, permit the following conclusions:

1. Splenoportography is usually successfully accomplished with very simple techniques, but it carries a certain percentage of failure and substandard opacification. Underlying causes and suggestions for correction are described.

2. Splenoportography is relatively free of major hazard considering the seriousness of the problems for which it is used. Bleeding requiring transfusion or splenectomy may be expected in about 2 per cent of the patients who undergo the procedure.

3. Splenoportography accurately indicates the presence of portal obstruction. Variations in technique do not influence portal hemodynamics. The site of obstruction is usually but not always identified.

4. Splenoportographic alterations due to hepatic and pancreatic neoplasm occur inconstantly. Reliance cannot be placed on negative examinations.

Department of Radiology
University Hospital
Ann Arbor, Michigan

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PERCUTANEOUS SPLENIC PORTOGRAPHY IN AMEBIC LIVER ABSCESS*

By TAWAN SURAWONGSE BUNNAG, M.D., VIRUL KAOPARISUTHI, M.D., SILLAWAT ARTHACHINTA, M.D., KALAYA CHIENPRADIT, M.D., *and* LUANG BINBAKAYA, M.D.
BANGKOK, THAILAND

PERCUTANEOUS splenic portography is, today, rather universally employed, and the literature contains much material on its usefulness in liver cirrhosis, carcinoma and other conditions, but so far no reports have appeared on its value in liver abscess.

We have performed splenoportography in amebic liver abscess for three reasons: (1) to demonstrate the intrahepatic portal venous pattern in this disease; (2) to locate the abscess for clinicians when such abscess could not be reached with the aspirating needle; and (3) as a follow-up examination after specific therapy.

This study is based on a series of 26 cases of liver abscesses, 5 of which were seen in 1956 and 21 in 1957.

ROENTGEN FEATURES

The common roentgen features which permitted differentiation from other diseases of the liver, such as cirrhosis and carcinoma were: (1) an enlarged liver the portal veins of which exhibited proportionate increase in size and length, at the same time retaining their soft smooth curves and gradual decrease in caliber, as long as cirrhosis did not intervene; (2) hypertrophy of the healthy liver tissue, most often of the left lobe, probably compensatory in nature; (3) an avascular area in the liver representing the area of the abscess. Here, the blood vessels were not displaced or crowded to one side, or stretched out as seen in a "space-occupying" lesion such as carcinoma; they were destroyed or occluded as the abscess was forming in the liver, thus being eliminated from the field; (4) absence of delay in opacification of the liver. Since there was no increased intrahepatic pressure, the sinusoids became opaque at the end of six to nine seconds; and (5) absence of abnormal reflux or varices, except when cirrhosis was also present (Table 1).

The site of the abscess in the liver can be located due to the fact that only the undamaged blood vessels are opacified, fewer vessels appear in the roentgenographic field and there is less superimposition of them as compared to those of the normal portogram. The courses of the veins can be followed and individual veins can often be identified. Since the missing ones belong to the necrotic liver segment, it is possible to give exact segmental location of the lesion in the liver by inference and by careful study of the opacified veins.

TABLE I

ANALYSIS OF PORTOGRAMS IN LIVER ABSCESS (20 CASES)

Common Features	No. of Cases	Remarks
Liver enlargement	19	
Left lobe hypertrophy	15	
Circulation time:		
slight delay	4	with cirrhosis
definite delay	5	with cirrhosis
normal	11	
Presence of cirrhosis	8	circulation time delayed
Presence of abnormal reflux	7	all with cirrhosis
Presence of avascular area	20	

Note: Only 20 cases of the series of 26 were analyzed. The remaining 6 cases were considered solely in follow-up studies after specific therapy.

* From the Department of Radiology, Chulalongkorn Hospital, Bangkok, Thailand. Read at the Ninth Pacific Science Congress held in Bangkok, Thailand, November, 1957.

Fig. 1. Opacified vein in the right lobe of the liver belongs to the posterior segment. It courses laterally, then curves upwards. Note the cephalad, lateral and caudad branches.

The case shown in Figures 1 and 2 illustrates an abscess cavity in the anterior segment of the right lobe of the liver. The patient was clinically diagnosed as having an amebic liver abscess, but no pus was obtained on aspiration. The splenic portogram (Fig. 1) shows, in the right lobe an opacified vein coursing laterally, forming a curve and giving off branches cephalad, caudad and laterally. These veins belong to the posterior segment of the right lobe. Those of the anterior segment are absent; therefore, the abscess is located anteriorly. After aspiration of the pus from the anterior superior pole of the right lobe, the portogram shown in Figure 2 was obtained. The abscess at this time appeared as a radiolucent area because some air had been

Fig. 2. Same case as shown in Figure 1 after aspiration of the pus from the anterior superior pole of the right lobe of the liver and introduction of air into the cavity. The abscess appears as a radiolucent area.

Fig. 3. The abscess cavity, a large radiolucent area in the liver, is superimposed by well defined veins of the anterior segment of the right lobe, not involved in the abscess formation. Note the branches extending medially, laterally, cephalad, and caudad. There is marked hypertrophy of the left lobe of the liver.

Fig. 4. Lateral projection of patient shown in Figure 3 confirms the posterior location of the abscess.

Fig. 5. A large avascular area in the right lobe of the liver, presumably a large abscess.

Fig. 7. Left lateral decubitus position showing lateral extension of the abscess. Same patient as in Figure 6.

introduced into the cavity after removal of the pus.

Figure 3 illustrates an abscess in the posterior segment of the right lobe of the liver. Pus was aspirated successfully, after which air was introduced and splenic portography was done to study the portal venous pattern in the liver. The abscess presents as a large radiolucent area on which are superimposed well opacified veins. Note the smooth curves retained by the veins. These veins belong to the intact anterior segment of the right lobe, which, in the anteroposterior projection, is superimposed on the abscess cavity. Also note the well formed medial, lateral, cephalad and caudad branches. The left lobe of the liver is enlarged. Figure 4, in the lateral projection, confirms the posterior location of the abscess cavity. Location of the diseased liver segment in these two cases is a significant finding for it opens up a new

Fig. 6. Absence of contrast in the right lobe of the liver, absence of abnormal reflux and presence of marked hypertrophy of the left lobe.

Fig. 8. Right lateral decubitus position showing medial extension of the abscess cavity. Same patient as in Figures 6 and 7.

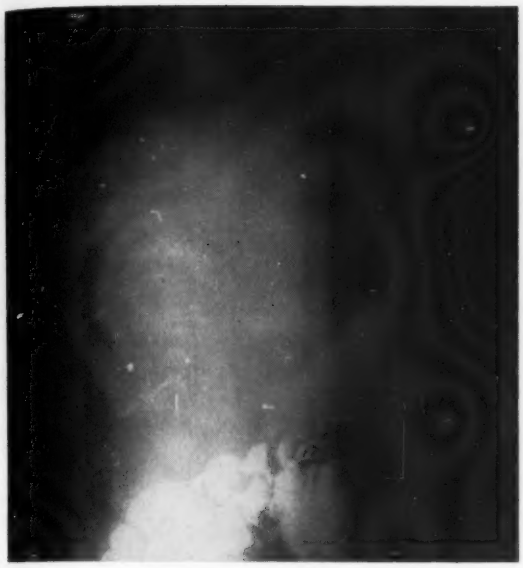

Fig. 9. Lateral prone position showing extension of the abscess anteriorly. Same patient as in Figures 6, 7 and 8.

Fig. 11. The normal blood vessel pattern in the right lobe of the liver is restored. No cavity is seen. There is marked reduction in the size of the liver three weeks after specific therapy. Same case as in Figure 10.

means by which the exact site of a lesion, other than an abscess which may require surgical removal, might be located.

We have seen fairly constant courses of the main portal veins in the right lobe of the liver. Those of the left lobe are more variable. So far, we have been able to identify segments of the right and left umbilical veins and branches to the superolateral aspect of the left lobe of the liver (Fig. 13). We realize that our present study is inadequate for identification of subsegmental veins and that more extensive study of the normal distribution of the intrahepatic portal venous system is necessary for proper

Fig. 10. A large cavity is seen in the right lobe of the liver with a fluid level. Note the hypertrophy of the left lobe, the negligible reflux, the soft curves and smooth outlines of the remaining blood vessels, and the posterior segment veins.

Fig. 12. Large abscess cavity in the right lobe of the liver, hypertrophy of the left lobe including hypertrophy of its right umbilical segment. Note the well opacified umbilical segmental veins.

FIG. 13. The cavity is almost invisible three weeks after specific therapy. Note the veins to the superolateral aspect of the left lobe and the marked decrease in the size of the liver. Same case as in Figure 12.

evaluation of the site of the affected area.

The cases shown in Figures 5 and 6 illustrate how extensive the necrosis in liver abscess can be. Figure 5 demonstrates a large avascular area in the right lobe, presumably occupied by the abscess. Note the marked hypertrophy of the left lobe and the absence of collaterals. Figure 6 reveals absence of contrast in the entire right lobe of the liver, resembling an obstruction produced by a mass; however, there are no refluxes. Figures 7, 8 and 9 show the same liver after aspiration of the pus and introduction of air in left lateral decubitus, right lateral decubitus and lateral prone positions, respectively. The extent of the abscess cavity conforms with the avascular area in the right lobe.

Figures 10 and 11 illustrate the remarkably rapid healing of an amebic liver abscess following specific therapy. Figure 10 demonstrates a large abscess cavity in the right lobe, before treatment, and Figure 11 discloses reorganization of the portal veins in the right lobe three weeks after the start of specific therapy. No abscess cavity is visible and the liver is very much reduced in size. Figure 12 is a similar case exhibiting all the common features of liver abscess, and Figure 13 shows the rapid healing after three weeks of specific therapy.

CONCLUSION

1. There is a pathognomonic pattern of the intrahepatic portal venous system in liver abscess; percutaneous splenic portography permits differentiation of this condition from other diseases of the liver.

2. Splenic portography is useful in assessing the progress made by proper treatment of liver abscesses.

3. Contrary to previous concept, amebic liver abscess regresses rapidly under specific therapy.

4. By proper recognition of the veins, it is possible to locate the site of the abscess in the liver.

SUMMARY

A study of the roentgen appearance of the portal venous system in 26 cases of liver abscesses has shown conclusive evidence that a definite portal venous pattern which differentiates the condition from other diseases of the liver exists, that regression of abscess cavities in the liver can be followed by means of percutaneous splenic portography, that liver abscesses heal rapidly under specific therapy and that percutaneous splenic portography may be employed to locate the site of the abscess in the liver. The need for further study of the normal distribution of the intrahepatic portal veins is expressed.

Tawan Surawongse Bunnag, M.D.
Department of Radiology
Chulalongkorn Hospital
Bangkok, Thailand

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ARTERIOGRAPHY IN NEOPLASMS OF EXTREMITIES*

By ALEXANDER R. MARGULIS, M.D., *and* THOMAS O. MURPHY, M.D.
MINNEAPOLIS, MINNESOTA

RECENT advances in the technique of peripheral arteriography have made this procedure one of the most valuable in the preoperative study and evaluation of neoplasms of bone and soft tissues of the extremities. It is a relatively simple and harmless examination that, since the introduction of new nonirritating contrast media (renografin*), causes almost no pain and can be done on an outpatient basis. Yet as innocuous and uncomplicated as this method is, it permits the establishment of the presence of a neoplasm. It outlines its extent, which cannot be done by conventional roentgenography. It may unmask unsuspected satellite lesions thus offering information on the basic nature of the process. It allows, in most instances, differentiation between an inflammatory lesion, a cyst or a tumor of bone and soft tissues, as well as between a benign and malignant tumor. It permits the *in situ* study of the vascular architecture of a neoplasm, and by the use of serial exposures it makes possible the understanding of the dynamics of circulation in the tumor itself and in the surrounding tissues. By arteriography one can also evaluate the effect that a neoplasm exerts on the circulation of the extremity as a whole. It affords information to the surgeon on technical problems of the area of biopsy and if roentgen therapy is chosen as the preferred treatment, it serves as an objective control of the progress and success of therapy.

It is surprising that a method that has so much to offer has not achieved popularity in the United States. This is probably due to the general belief that peripheral arteriography is fraught with dangers and its use is only valuable in the study of occlusions of major vessels. The opinion that arteriography does not offer any advantages over routine roentgenographic studies is shared by many radiologists in this country.

Since dos Santos introduced peripheral arteriography in the study of bone tumors, this method has produced considerable interest in Europe. The lack of suitable contrast media has prevented its wide usage, yet by 1950 dos Santos[4] was able to report his experiences based on the study of 100 tumors. Since then arteriography has become quite a popular procedure in Europe and has been extensively employed in the study of tumors of bone and soft tissues. The communications of Borgström,[1] Caldas,[2] Mucchi and Columella,[8] Denstad,[3] dos Santos,[4] Fariñas,[5] Pape and Seyss,[10] Sutton,[13] Scholz,[11] Vogler and Deu,[14] and the review by Wellauer[15] are indicative of this extensive interest. The degree of enthusiasm for the procedure by some of these authors is reflected in the concluding statement of the article by Mucchi and Columella:[8] "The appearances in malignant tumors are so clear-cut and characteristic that confirmation of the diagnosis by biopsy can be dispensed with."

The purpose of this study is to report on our experiences with this method, present our findings, evaluate the indications and discuss the limitations.

TECHNIQUE

The technique of peripheral arteriography has been reported previously.[9] Thirty-five to forty per cent renografin was the contrast agent of choice. The following observations were made: (1) Little pain was experienced by the patients; (2) no anesthetic other than local procaine was necessary; (3) the material was quite safe and free from side effects, so that the pro-

* Squibb.

* From the Departments of Radiology and Surgery, University of Minnesota Medical School, Minneapolis, Minnesota. Presented at the Fifty-eighth Annual Meeting of the American Roentgen Ray Society, Washington, D. C., October 1–4, 1957.

cedure could be often performed on an outpatient basis; (4) the opacification of vessels and detail were satisfactory and compared favorably with other media.

The injection was routinely made into the femoral artery, by the percutaneous route with the medium directed proximally. The tunnel box devised by Watson and Nelson and described elsewhere[9] was used in the first study. By this technique, both an early and late arterial phase were visualized making the study of any arteriovenous shunts and open bypasses possible. If more information about the flow within the neoplasm was desired, the area selected was studied by means of a repeated injection using the Watson-Rigler serial changer with one to four exposures per second. A few cases have also been studied by means of cineroentgenography. Both latter methods were found to show only a relatively small area, thus limiting the information to the area of tumor only. No information concerning possible satellite lesions or effect of the tumor on the remainder of the extremity is obtained.

In order to procure studies of the whole extremity using one exposure, the "heel" effect was directed toward the thigh and the single cassette employed longitudinally for the two exposures contained two types of intensifying screens—very fast screens of low detail over the upper thigh and slow-high detail screens over the rest of the extremity. This compensated for the greater thickness of the thigh.

MATERIAL

Four cases of osteogenic sarcoma were examined, as well as 3 cases of multiple myeloma, 4 cases of fibrosarcoma, several cases of neurofibrosarcoma, Kaposi's sarcoma, chondrosarcoma, fibrous dysplasia with malignant degeneration and a multitude of metastatic bone lesions including lesions of the breast, bronchial carcinoma metastases and metastatic melanosarcoma among others. Among benign lesions an endothelial fibroma was studied as well as inflammatory conditions of bone and soft tissues, synovial cysts, Paget's disease, recent fractures, hematomas and fibrous dysplasia. All cases had one study of the whole extremity and some had the study repeated utilizing the serial changer and/or cineroentgenography.

APPEARANCE ON ARTERIOGRAPHY

The study contained too limited a number of cases of each condition to be able to draw conclusions concerning a characteristic vascular pattern of any given lesion. Unfortunately, this is also true of all available reports in the literature with the exception of the article by dos Santos.[4] He reports that giant cell tumors show an identifiable pattern, with pools of contrast material and rather slow filling of the venous tree. It is possible that, if enough experience were available for other neoplasms, a method close to histologic accuracy could evolve.

Most malignant tumors are very vascular while most benign tumors are relatively avascular. This especially holds true in instances of bone neoplasms, but is also applicable for tumors of soft tissues. Vogler and Deu,[14] however, pointed out that there are some malignant avascular tumors as well as vascular benign neoplasms. Dos Santos believed the rapidity of circulation through a tumor and the early filling of veins to be a sign and even a measure of malignancy. This we found not to be completely true (Fig. 8). One of our fibrosarcomas with widespread pulmonary metastases was indistinguishable by arteriography from synovial cysts (Fig. 9). Conversely, we have had a case of aseptic soft tissue necrosis that had the arteriographic appearance of a malignant tumor with massive arteriovenous shunts, irregular vessels, pooling of the contrast material and very rapid circulatory flow through the area of involvement. This was not reported by other writers in this field. Caldas,[2] dos Santos[4] and others have made statements that such appearance is an accurate sign of malignancy.

In describing the arteriographic appearance of tumors of extremities it is easier to

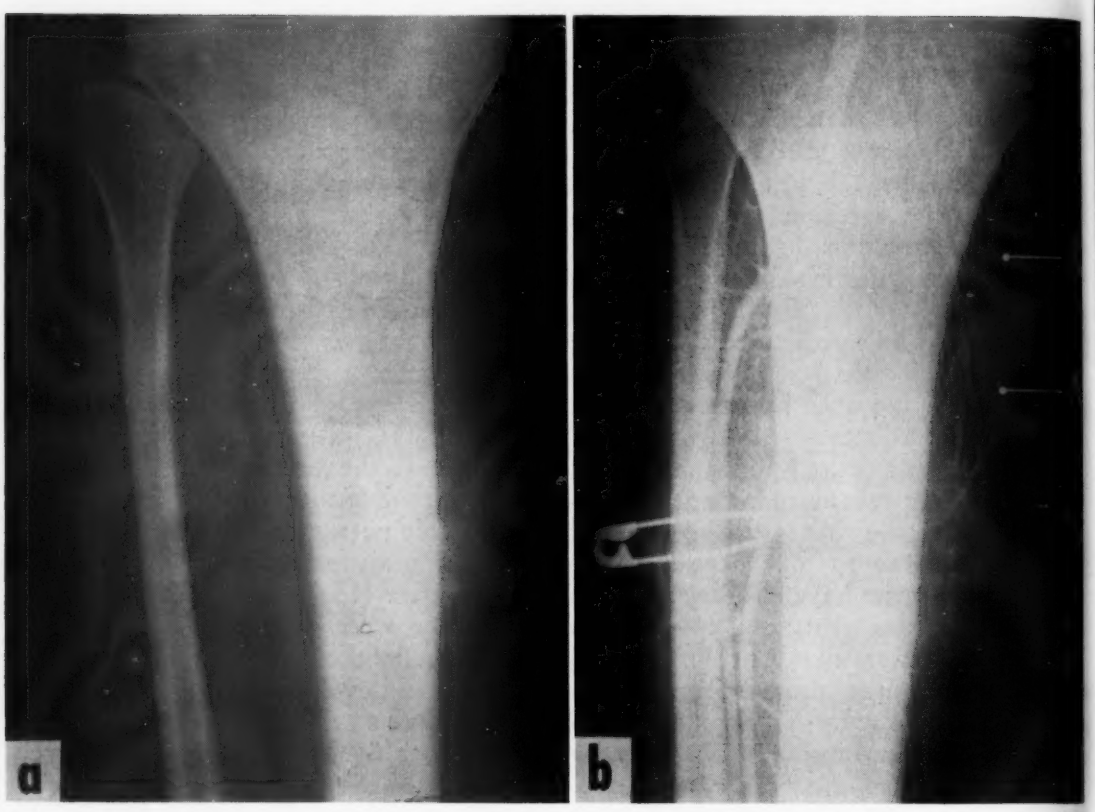

Fig. 1. Osteogenic sarcoma. (*a*) Plain roentgenogram showing new bone formation within the soft tissue mass. (*b*) Arteriogram. Large veins (A) are seen simultaneously with arteries indicating arteriovenous shunts within the tumor. An irregular network of abnormal vessels is noted within the neoplasm with tumor stain. There are small superficial arteriovenous communications present in the soft tissues lateral to the fibula.

divide them into tumors of bone and those of the soft tissues.

BONE TUMORS

In all primary and metastatic bone tumors in our series as well as those reported in the literature, the malignant varieties showed a characteristic pattern. Large arteries are seen supplying the tumor and almost immediately large veins are noted draining the tumor area. The shunts within the tumor are massive and the large veins are densely opacified. This is true of both primary and secondary malignancy. The amount of shunt and its abundance, however, is not a good indicator of the degree of malignancy as even some histologically very malignant osteogenic sarcomas may show relatively unimpressive shunts (Fig. 1). Several authors, as for example Caldas[2] and Wellauer,[15] have expressed the opinion that malignant bone tumors show abnormal vascularity arteriographically only after the bone tumor establishes secondary vascular connections with dependence upon the arteries from adjacent soft tissue and is deriving its supply entirely from the nutrient arteries of bone. In all of our cases the tumor showed a vascular supply from the adjacent soft tissues.

All of the bone tumors studied and their extension to the soft tissues revealed an irregular network of vascular channels, with localized areas of widening of lumen. The extent of the irregular network as pointed out by many authors is an accurate outline of the extent of the tumor, a much

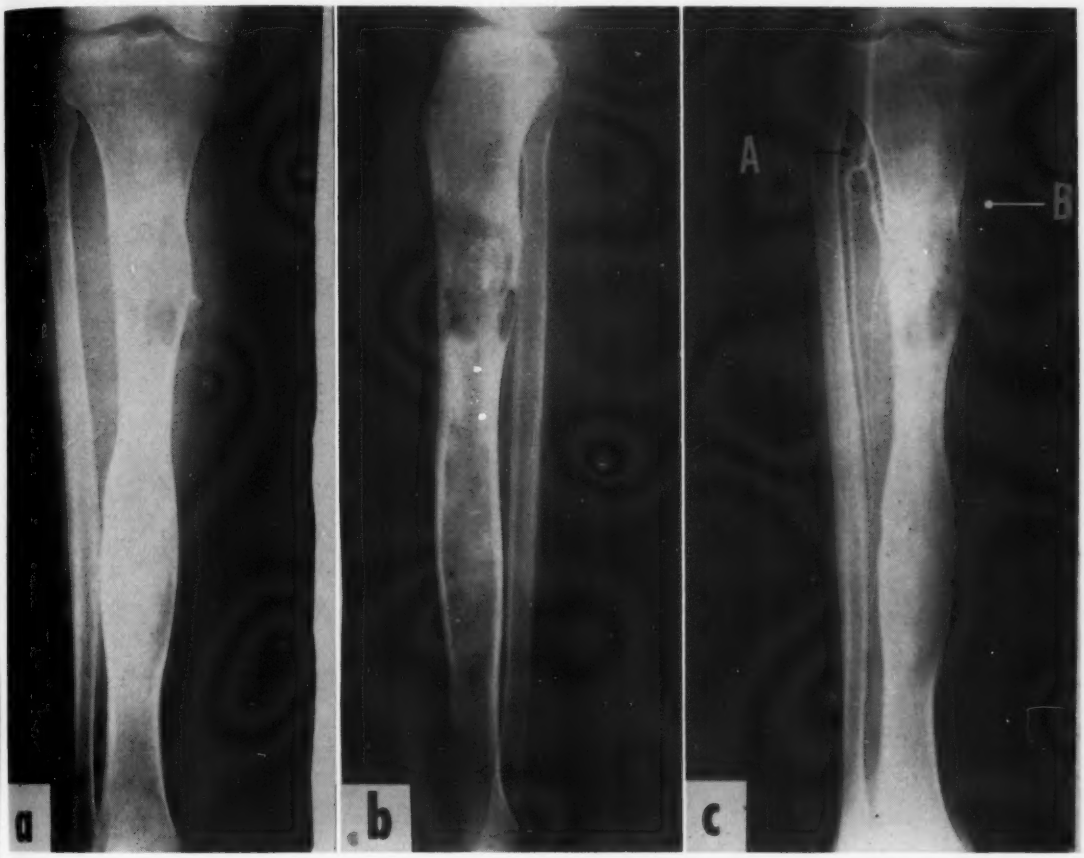

Fig. 2. Fibrous dysplasia with malignant degeneration into fibrosarcoma of the right tibia. (*a*) Plain roentgenogram in frontal projection. (*b*) Lateral view. (*c*) Arteriogram. Irregular vessels are seen in the area of the tumor extending into the soft tissues. (A) Arteriovenous communications outside the tumor. (B) Veins carrying blood out of tumor are visualized simultaneously with the arteries.

more sensitive index than either the naked eye or the routine plain roentgenogram.

In addition to the vascular changes within the tumor itself, and contrary to the reports of other workers, all of the malignant bone lesions that we studied showed evidence of multiple small arteriovenous shunts elsewhere outside the tumor area and even distal to it. We have seen both rete and "H formations," as well as secondary signs of arteriovenous communications abundantly in localized areas (Fig. 1, 2 and 4). It was proved by dissection of the specimen that no malignancy was present in the areas illustrated. The etiology of these shunts is not clear. They may represent arteriovenous bypasses that are normally present but which have escaped neuromuscular control.[12] Sir Thomas Lewis[6] has shown that long standing massive arteriovenous communications will, through some unknown mechanism, produce massive openings of the normal arteriovenous bypasses. This will result in increased volume of the extremity distal to the site of this massive shunt and will give signs of arteriovenous communications distally. We have also been able to demonstrate this by arteriography in traumatic arteriovenous fistulae and in arteriovenous communications of congenital or arteriosclerotic origin. It is likely that this same mechanism produced by a massive localized arteriovenous shunt in the tumor causes the opening of the normal arteriovenous shunts elsewhere. The implications of such

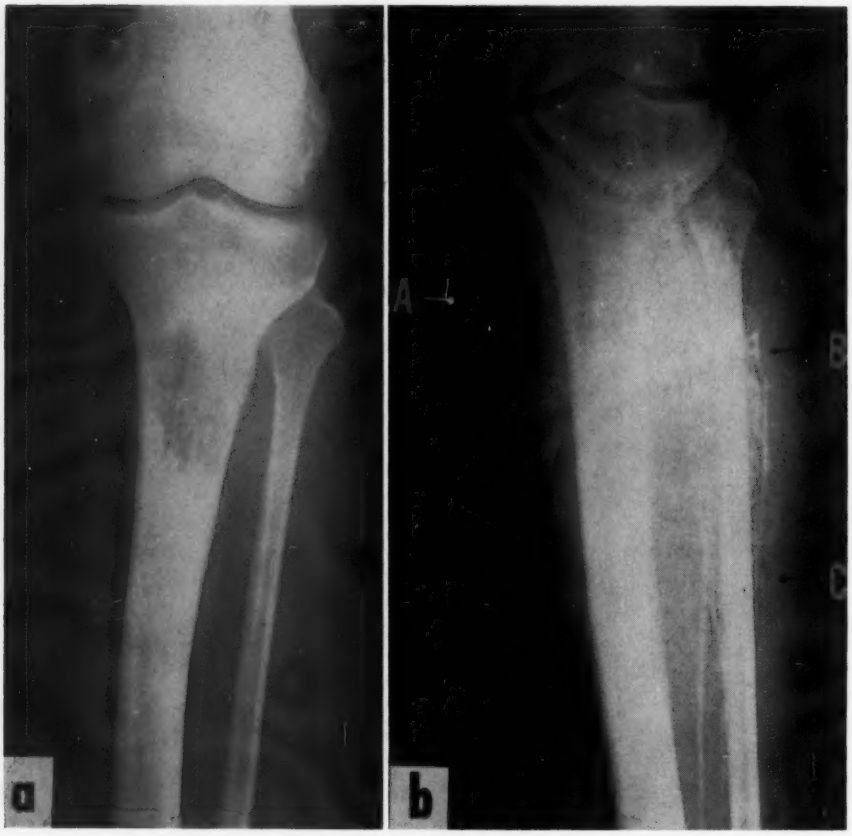

Fig. 3. Metastatic carcinoma of the breast in left tibia. (*a*) Plain roentgenogram shows a destructive lesion in the proximal tibia with periosteal elevation and soft tissue extension. (*b*) Arteriogram. (A) Large veins are seen in the soft tissues indicating a massive arteriovenous shunt. (B) Superficial veins in the region of the tumor are visualized simultaneously with arteries. (C) Simultaneous demonstration of veins distal to the tumor supplied by arteriovenous bypasses.

increased speed of blood flow through the extremity as a whole are obvious. It is interesting that even a case with aseptic muscle necrosis that had the arteriographic appearance of a malignant tumor of the soft tissues had extensive distal arteriovenous communications.

As inflammatory lesions of bone do not show any abnormal vascularity, arteriography can play an important role in differentiating such lesions from tumors, which frequently cannot be done by plain roentgenography. Such lesions as fibrous dysplasia, calcified hematomas and fractures will not show any abnormal vessels. In our few cases of Paget's disease studied by arteriography, the findings were the same as those described by Caldas,[2] namely, an increase in the number of vessels in the soft tissues adjacent to the bone, some of the vessels being most likely periosteal. We also found vessels joining these periosteal ones at 90 degree angles, creating structures similar to the "H formations" of arteriovenous communications.

SOFT TISSUE TUMORS

A. *Malignant Tumors with Increased Vascularity.* The majority of malignant tumors studied belong to this group. In general, such tumors showed a vascularity similar to that of malignant bone tumors. An irregular network of vessels of varying caliber and irregular outline is characteristic. Quite frequently areas of tumor are seen which showed a tumor stain. This is a solid uniform increase in density undoubtedly caused by simultaneous filling of many

very small vessels by the contrast material. Even in the very vascular tumors, when large, there were areas showing little or no vascularity (Fig. 6). Some tumors as demonstrated by a case of Kaposi's sarcoma (Fig. 7) showed no network of abnormal vessels but only solid tumor stain indicating that all the vessels filled were small and quite numerous. In that particular case a cineroentgenographic study showed the artery supplying the vascular lesion and the vein draining it almost simultaneously. The artery and vein are also seen simultaneously on the routine arteriogram. While our experience is limited and does not warrant definite conclusions, we have found that fibrosarcomas of soft tissue, even the ones showing a great deal of vascularity, will not exhibit massive filling of veins. While this may mean that the arteriovenous shunts within such a tumor are not as marked as in other malignant tumors, there is a possibility that there is pooling and lag in the tumor vascular bed. The demarcation of tumor from the remainder of the extremity was quite sharp in the tumors with increased vascularity. We have also noted in our fibrosarcomas of the soft tissues, a lack of arteriovenous shunts outside the tumor, while other soft tissue malignancies had them. Some of our cases of fibrosarcoma also showed simultaneously areas of "tumor stain" and the network of irregular vessels (Fig. 5).

B. *Malignant Tumors with Decreased Vascularity.* Vogler and Deu[14] first called attention to the possibility of a malignant tumor being avascular. They had 14 such cases, a relatively large proportion in their series. The majority of their cases were soft tissue sarcomas (8 cases) but they also had 3 sarcomas of bone and 2 metastatic bone lesions that were avascular. They pointed out that it is impossible on the basis of such studies to tell whether a lesion is malignant, although in some instances conclusions may be drawn from the irregular outline of the avascular area. In these cases, according to Vogler and Deu,[14] the normal vessels are not as displaced as in benign growths due to the infiltrative nature of the malignancy. In the capillary phase such tumors will show a cloudy irregular staining. We had only one study upon a fibrosarcoma with multiple pulmonary metastases which showed little vascularity but displaced the normal vessels in the manner encountered by cysts (Fig. 8).

FIG. 4. Metastatic melanosarcoma in right tibia. (A) Superficial veins indicative of arteriovenous shunts within the tumor. (B) Superficial shunts outside the tumor area.

C. *Benign Avascular Tumors.* Most benign avascular tumors are cysts although lipomas, myxomas, and hemato-

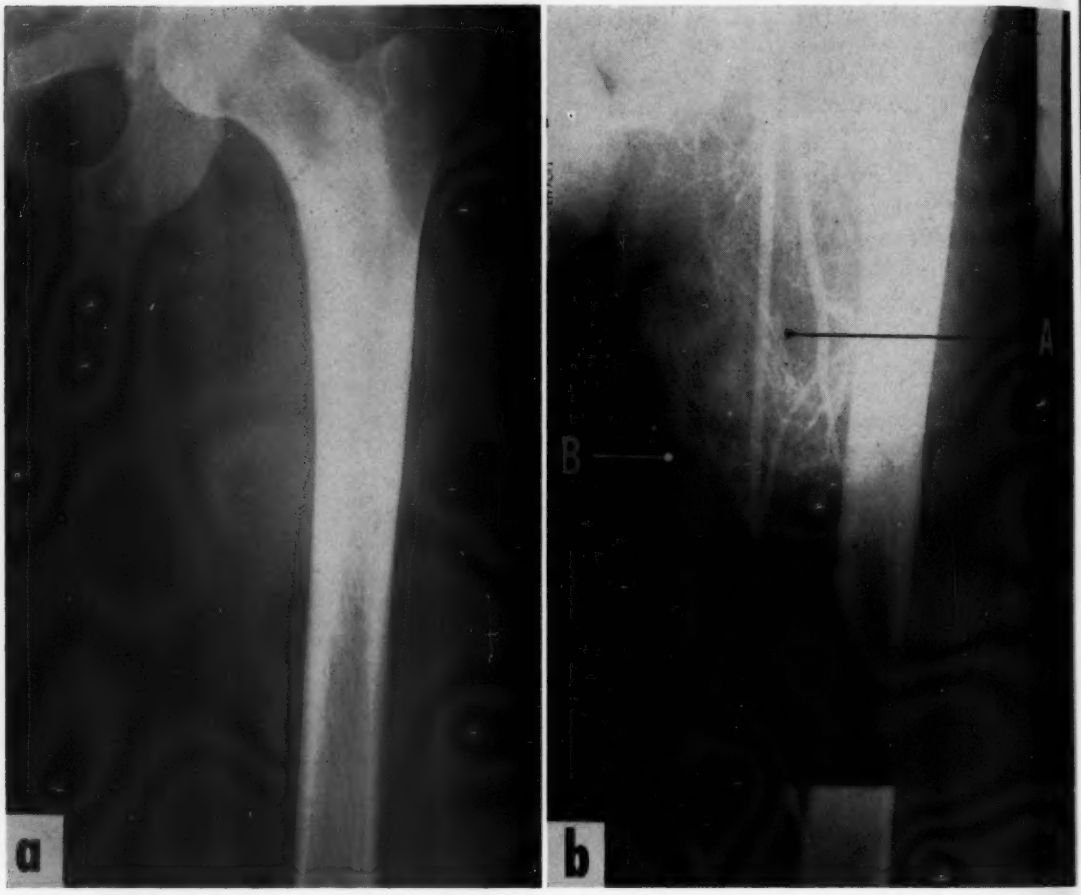

Fig. 5. Fibrosarcoma of the left thigh. (*a*) Plain roentgenogram shows a large soft tissue mass. (*b*) Arteriogram shows a network of irregular, abnormal vessels; veins visualized in the soft tissues (A) are not abundant. There is tumor stain (B) of lobules of the tumor.

mas will produce an indistinguishable appearance (Fig. 9). There is good outline of the tumor with the normal vessels displaced and following a smooth course over the surface of the tumor, which often is spherical. No abnormal arteriovenous communications are visualized either within the tumor or within the rest of the extremity.

D. *Benign Vascular Tumors.* The majority of such tumors are vascular hematomas.

Fig. 6. Neurofibrosarcoma of the left thigh. (*a*) Arteriogram shows the early arterial phase and (*b*) the late arterial phase. Abundant irregular tumor vessels are seen in some areas. There is a lack of demonstrable large veins.

FIG. 7. Kaposi's sarcoma of right thigh. (*a*) Plain roentgenogram. No calcification is seen. (*b*) Arteriogram. (A and C) Highly vascular round lesions are imbedded in the soft tissues. The upper lesion (A) was totally unsuspected clinically. (B) Catheter.

In one case of endotheliofibroma of the thigh in a young girl, there was a relatively small area rich in small, fairly regular vessels. No veins were demonstrated simultaneously with the arteries. No irregular "tumor stain" was seen. These must be differentiated from congenital arteriovenous communications. The practical value of such studies is in the surgical management of their blood supply.

ACCURACY OF DIAGNOSIS

It is impossible to derive significant statistical conclusions on the basis of our rather limited experience, as well as on the basis of most reports to date. We have felt quite confident of our diagnosis of malignant bone tumors, all of which showed increased vascularity, and found that in all of these studies our opinion based on arteriography was more accurate than either the opinion based on the plain roentgenograms or even the surgical impression at operation. In one case, where frozen section histopathologic diagnosis was made, the arteriographic interpretation was more accurate. As far as soft tissue lesions were concerned when abnormal vascularity was encountered, the opinion as to malignancy was quite reliable. The one case with aseptic muscle necrosis was felt to be an exception, and is undoubtedly rare since there is no other report in the literature about a similar case. Some members of our staff believe that, in spite of repeated biopsies in this case, it cannot definitely be declared a failure of arteriography until the pathologic diagnosis of inflammatory disease has withstood the test of time. We are quite aware of multiple reports in the literature of hyperemia and increased vascularity in cases of active inflammatory processes;

Fig. 8. Fibrosarcoma of the right thigh in a patient with multiple pulmonary metastases. The lesion is located behind the distal femur. Normal vessels are noted to be displaced by the soft tissue mass and surround it. No abnormal vessels or arteriovenous communications are seen.

however, as Vogler and Deu,[14] and Caldas[2] point out, one deals in such cases with normal but increased vasculature, and in instances of abscess, with displacements of such normal vessels. These cases do not show the abnormal network and the arteriovenous shunts in the area of involvement such as is characteristic of tumors.

In the instances of avascular soft tissue tumors, the diagnosis is much less reliable and one has difficulty excluding malignancy. Fortunately, cases as the one presented in Figure 8 are rare. In addition, it is to be stressed that, as with other roentgen studies, arteriographic interpretation should also have the benefit of clinical information, and pertinent features from roentgenograms should be brought to bear on the final opinion in equivocal cases. Rapid growth of a mass, presence of metastases elsewhere, loss of weight, anemia, etc. are too important data to be disregarded.

DISCUSSION

It is to be emphaszied that in spite of some minor pitfalls, arteriography is generally reliable in the differential diagnosis between inflammatory lesions and malig-

Fig. 9. Synovial cysts. Large soft tissue masses in front and behind the knee. Normal vessels are displaced. There are no abnormal vessels and no arteriovenous communications.

nant tumors of bone. The contribution of dos Santos[4] in pointing out the difference between Ewing's tumor and osteomyelitis is by itself a triumph for the procedure. In some obvious instances of osteogenic sarcoma, arteriography may not be of great value in establishing the diagnosis. Nevertheless, one obtains information concerning the degree of extension of the tumor into the soft tissues, about the vascular shunt within the tumor, and the influence of the tumor upon the vascular status of the rest of the extremity. Occasionally, satellite lesions become obvious, indicating that one is dealing with metastases or with multiple myeloma. This is especially true in instances of soft tissue lesions (Fig. 7). Benign lesions, such as fibrous dysplasia and Paget's disease, may show some change which is suspicious of malignant degeneration, thus assisting at biopsy. These conditions are puzzling and variable; however, if an arteriogram shows evidence of malignant changes, a more positive diagnosis can be made.

In tumors of the soft tissues, arteriography can prove a valuable procedure, especially in view of the reluctance to resort to biopsy.

SUMMARY

Experience with arteriography in the study of neoplasms is presented. The method is harmless and the procedure can be performed on an outpatient basis. In most cases the presence of a tumor can be established. The differentiation between a malignant and benign lesion can be made. The outline of the neoplasm can be obtained. The vascularity of the area is studied concomitantly with the effect of the tumor on the circulation of the rest of the extremity.

Alexander R. Margulis, M.D.
University of Minnesota Medical School
Minneapolis 14, Minnesota

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TECHNICAL AIDS IN CEREBRAL ANGIOGRAPHY*

By FRED C. SHIPPS, M.D.,† *and* JOHN RAAF, M.D.‡
PORTLAND, OREGON

THIS article concerns the roentgenologic problems encountered in cerebral angiography. Our recent technical adaptations herein described have attempted to accomplish the following: (1) more consistently filled arterial phases; (2) reduced procedural and anesthetic time; (3) reduced total contrast material dosage; and (4) reduced team tension.

PAST METHOD

In brief, this consisted of using 10–12 cc. of 35 per cent diodrast injected over one to two seconds for internal carotid arteriography. Near or at the end of injection the surgeon used the word "now" as the signal for the technician to begin the roentgenographic exposure. We routinely obtained single exposures in the anteroposterior and lateral views, and also six lateral serialograms with a Sanchez-Perez device. Occasionally, oblique views or slightly oblique lateral views were found necessary to substantiate or rule out a small aneurysm.

PRESENT METHOD

Our present method utilizes a syringe containing an electrical switch. This is connected to a delay timer which in turn activates the roentgen-ray control. The surgeon is relieved of any attention to timing factors. His only thought after filling the syringe is of the *rate* of injection.

Syringe. The syringe as shown in Figure 1 is a slight modification of a syringe described by Flax and Waldron[4] for use in aortography. It is easily constructed by modifying a standard B-D control syringe as Flax and Waldron describe in their paper. The contractor of the syringe is shockproof. It activates only a 6-volt circuit and withstands autoclaving in the surgical pack. We used Celeron plastic for the insulating material.

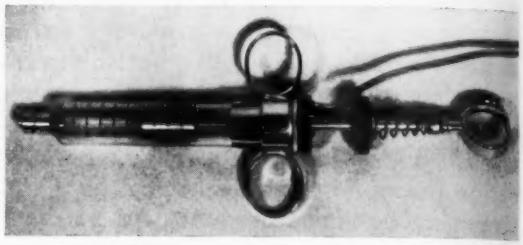

FIG. 1. Modified Flax and Waldron syringe.

Our modification of Flax and Waldron's design is the addition of a spring between the contractor and the upper end of the plunger so that the electric circuit would close 2.5 cc. before the end of injection. This permits an exposure before the end of injection if desired. This is useful in cases of arteriovenous fistulas where the carotid artery empties very rapidly.

Delay Timer. The function of the delay timer (Fig. 2) is to delay the roentgenographic exposure a pre-set interval. It is situated outside the sterile field. There are

FIG. 2. Delay timer.

* Presented at the meeting of the Academy of Neurological Surgery, Phoenix, Arizona, November 8–10, 1956.
† Department of Radiology, Good Samaritan Hospital, Portland, Oregon.
‡ Department of Neurosurgery, Good Samaritan Hospital, Portland, Oregon.

two multipole switches in the center of the panel. The upper switch is graduated from zero to ten seconds at 1.0 second intervals. The lower switch has settings of 0, 0.25 sec., 0.50 sec., and 0.75 sec. By combining settings, any quarter second delay up to 10.75 seconds can be obtained. Obviously, the longer delays are used in other types of angiography. When the timer is activated by the syringe and the proper delay has occurred, a red signal lights on the left side of the panel and the roentgenographic exposure begins. A push button on the right will deactivate the timer for further use.

During the final preparation of this paper, the authors discovered that the combined use of an electric syringe and delay timer in cerebral angiography had been previously reported[3,9] as a forerunner to the serialographic devices.

COMPARISON OF OUR PAST AND PRESENT RESULTS

In order to more objectively analyze our results we devised the system of grading arterial phases as shown in the illustrated code of Figure 3. Group 3 represents the most desirable arterial phase, and all groups above and below are progressively less desirable. It is evident that the percentage of ideal arteriograms rose appreciably, *i.e.*, from 58 per cent to 89 per cent. With the present technique those cases found in Groups 2 and 4 represent patient factors in rate of cerebral circulation.

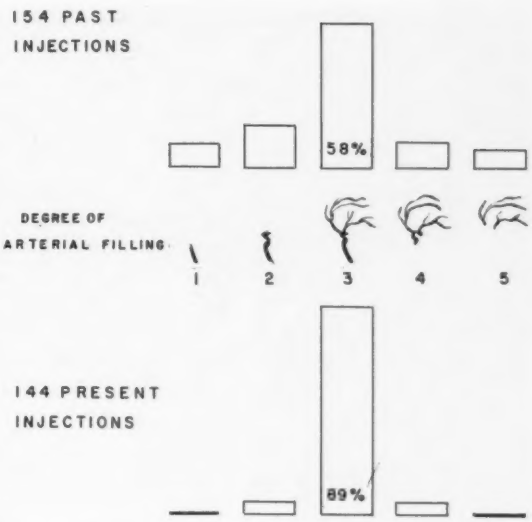

FIG. 3. Grading of arterial filling and comparison of results of past and present cerebral angiographies.

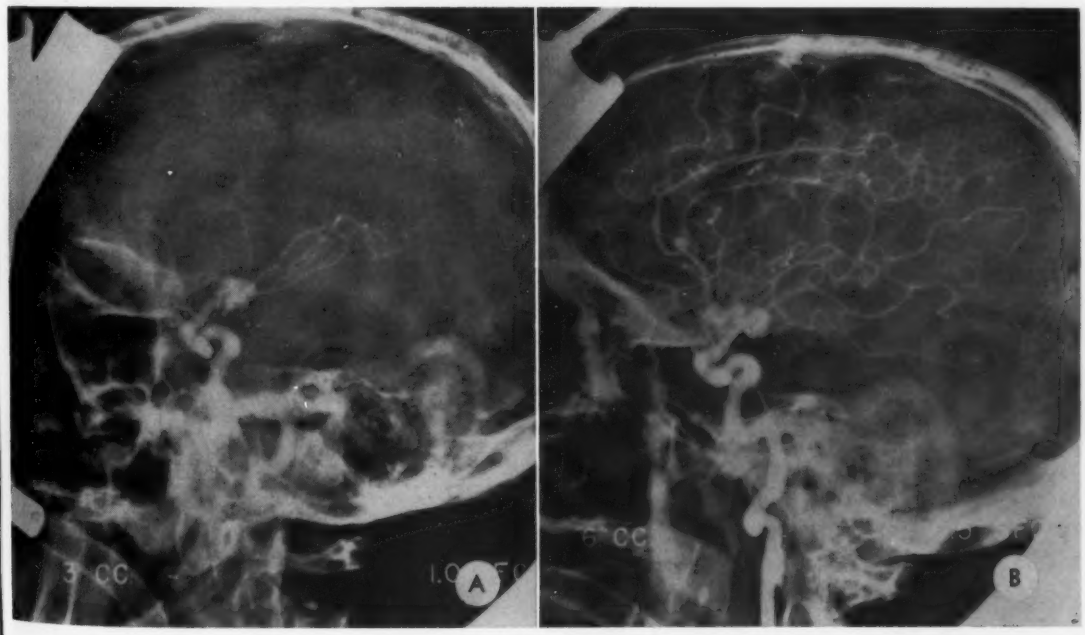

FIG. 4. (*A*) Arteriogram using 3 cc. hypaque, 1.0 sec. delay. (*B*) Arteriogram using 6 cc. hypaque, 0.5 sec. delay. Four aneurysms.

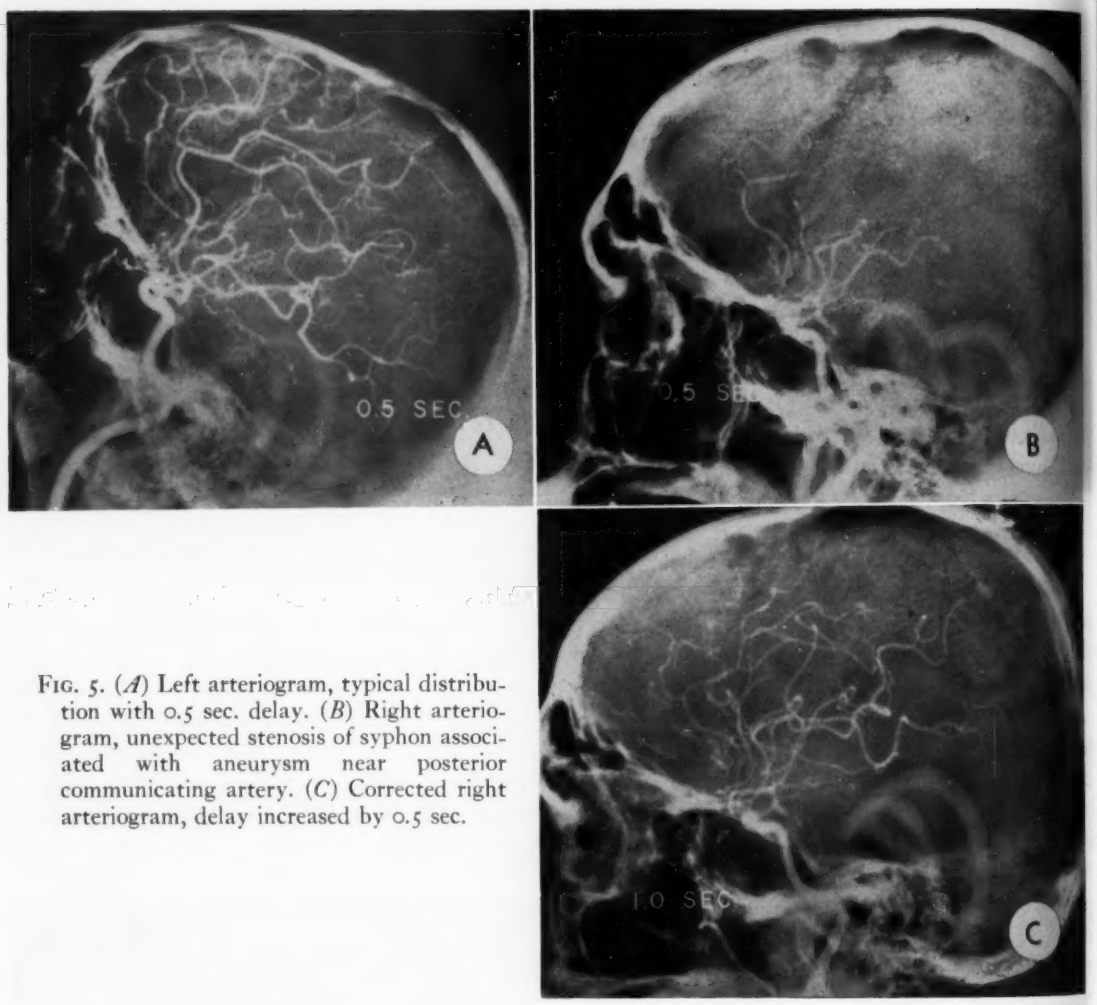

Fig. 5. (*A*) Left arteriogram, typical distribution with 0.5 sec. delay. (*B*) Right arteriogram, unexpected stenosis of syphon associated with aneurysm near posterior communicating artery. (*C*) Corrected right arteriogram, delay increased by 0.5 sec.

FACTORS IN OBTAINING THE ARTERIAL PHASE

Our first objective with these devices was to obtain a consistently good arterial phase from each injection and in every patient. There are several factors affecting the results. Though interrelated, they may be discussed separately.

a. *Volume and rate of injection.* Various authors[2,3,6,7,8] advise from 5 cc. to 15 cc. of diodrast. Their recommendations on rate of injection vary from "slow" to "as fast as possible." While using 35 per cent diodrast, we were able to reduce the volume from 10–12 cc. to 8 cc. without noticeable loss of sharpness. The rate of injection is still manually determined by the surgeon who tries to complete the injection in one second. Recently we have used 50 per cent hypaque. Aliquots of various sizes were injected in one second and we found that 4 cc. produces a shorter opaque column than we routinely desire. We use 6 cc. of hypaque for initial lateral roentgenograms and reduce to 4 cc. if attention is centered on aneurysms in the vicinity of the internal carotid artery.

b. *Instant of exposure.* It has been generally stated that the film should be exposed at the end of injection or earlier.[2,3,5,6,7] We found that with large aliquots (10–12 cc.) the best arterial phase was usually 0.25 sec. delay on the delay timer. With small aliquots (6 cc.) the full

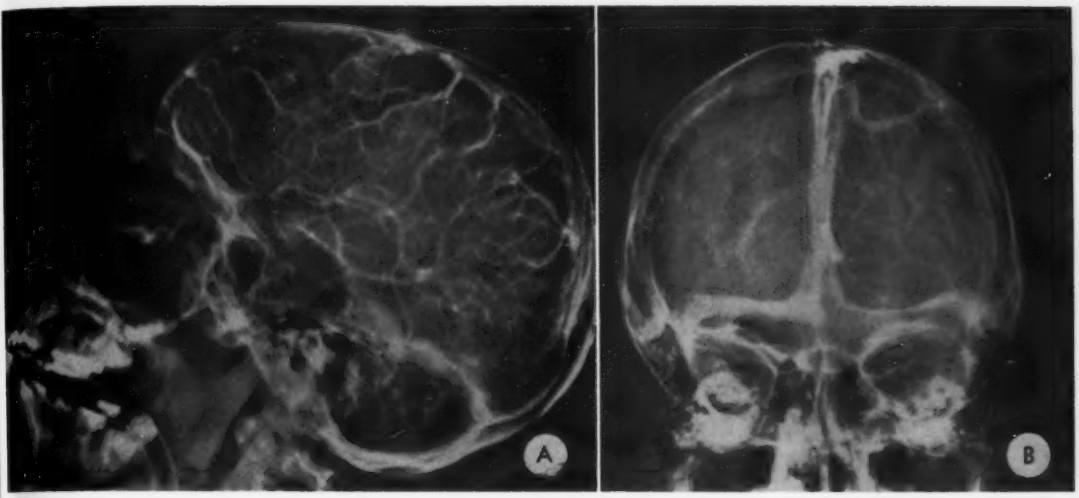

Fig. 6. (*A*) Lateral venogram, third exposure of serialogram. (*B*) Anteroposterior venogram, single exposure duplication of lateral view (see text).

arterial phase is caught at 0.5 sec. delay on the delay timer, and with 4 cc. we use 1.0 sec. delay (Fig. 4).

c. *Blood velocity*. Variation in the rate of blood flow is the greatest uncertainty in cerebral angiography. Correction of errors in time of exposure due to patient variations is the most common use of the delay timer. As previously stated, the optimal arterial phase is obtained by 0.5 sec. delay in the timer when 6 cc. of hypaque is injected in 1.0 sec. We routinely begin with this technique and inspect the first roentgenogram before proceeding with the remaining exposures. When a variation in blood velocity is encountered, changing the time by 0.25 sec. up to 1.0 sec. will insure perfect exposures of all subsequent films on the same side. Figure 5 illustrates a 0.5 sec. correction.

ANOTHER USE OF THE DELAY TIMER

Duplication of Serialographic Findings by the Single Exposure Technique. As previously mentioned, our procedure includes lateral serialograms using the Sanchez-Perez device. We have checked the time between exposures of the six consecutive films at maximum speed. Instead of 0.5 sec. intervals as indicated on the speed dial, we find intervals of 0.8, 0.8, 0.6, 0.6, and 0.6 seconds.

Although we feel that the Sanchez-Perez device produces as sharp a definition as any serialographic device commercially available, there are occasions when more definition is desirable in the capillary or venous phases. The single exposure technique with a grid cassette results in the best arteriograms that we can produce. If for instance we wish to reproduce the findings on the third film of the serialogram, we add $0.8+0.8=1.6$ sec. to the original delay time. Figure 6 illustrates the accuracy of reproduction and the improved quality of single exposure duplication. This system is an inexpensive substitution for the simultaneous exposure devices.

SUMMARY

Technical improvements in cerebral angiography have been presented using an electrical syringe with a variable delay exposure device.

Fred C. Shipps, M.D.
214 Medical Dental Building
Portland, Oregon

We wish to express our appreciation to Mr. Harold Brewer of Picker X-ray Corporation for work in both design and construction of the delay timer.

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EDITORIAL

INSTRUCTION COURSES OF THE FIFTY-NINTH ANNUAL MEETING OF THE AMERICAN ROENTGEN RAY SOCIETY

AT THE Fortieth Annual Meeting of the American Roentgen Ray Society held at the Palmer House, Chicago, Illinois, on September 19 to 22, 1939, Dr. Edward L. Jenkinson, President-Elect for that meeting "made a departure in the arrangement of the program" by introducing for the first time at meetings of the American Roentgen Ray Society the Instruction Courses, which were held in the afternoon, leaving the morning free for the scientific papers. Dr. B. R. Kirklin was appointed Director of the Instruction Courses and he had surrounded himself with a faculty of radiologists of outstanding ability and great authority. At once, these courses became so popular that the hope was expressed that "they will become a permanent feature of the Society's Annual Meetings."

Dr. Jenkinson, who with great foresight inaugurated this "departure in the arrangement of the program," has since then been amply rewarded by the continuously growing importance of these courses. Year after year, the President-Elects of the Society, who have succeeded each other, not only maintained the original innovation but endeavored to enlarge its scope so as to encompass the newest contributions to the science of radiology. The Directorship of the courses was entrusted to Dr. B. R. Kirklin, later to Dr. Harry M. Weber, and in 1957 to Dr. Harold O. Peterson, who with their enthusiasm made special efforts to meet the challenge and at all times obtained remarkable successes.

Dr. Harold O. Peterson, the Director of the Instruction Courses of the Fifty-ninth Annual Meeting, which will be held at the Shoreham Hotel, Washington, D. C., September 30 to October 3, has assembled a faculty for the forthcoming meeting which is the largest in the Society's history and composed of most distinguished members.

The wide array of subjects chosen by the faculty members for their lectures is likewise of most outstanding quality.

A comparison of the topics of the first Instruction Courses in 1939 and of the present ones, nearly twenty years later, depicts more vividly than words can express the tremendous progress that has been made in the science of radiology during this interval. A commensurate advance in the teaching and dissemination of the new achievements accomplished inevitably became necessary and had indeed become an essential feature of the program of the Annual Meetings of the American Roentgen Ray Society.

A brief glance over the Instruction Courses given in 1939 shows that about two-thirds of the lectures covered various phases of diagnostic roentgenology and the remainder were devoted to therapeutic radiology. Among the latter were included several courses in radiation physics dealing mostly with dosimetry and techniques, which were the important contributions to the science of radiology of that period. A few lectures were also given on anatomy, physiology and pathology, as related mostly to diagnostic roentgenology.

The experiences gained in World War II, the event of the atomic explosions in Hiroshima and Nagasaki, followed by the many ramifications of the medical use of atomic

energy and the unexpected technology resulting from this use, have opened for the scientific radiologist new avenues of knowledge of inestimable scope. The subjects chosen for this year's Instruction Courses abundantly reflect the thought given by the members of the Faculty to these new demands upon the scientific radiologist. There will be 64 courses by 57 instructors. Forty-four courses will be given in diagnostic roentgenology and 20 in therapeutic radiology, nuclear medicine, physics and radiobiology. The courses in diagnostic roentgenology include 29 single period courses, 6 two period courses and 1 three period course. The courses in therapy include 14 single period courses and 3 two period courses. Among the courses in therapy are 1 in chemotherapy, 2 in nuclear medicine, 1 in physics and 2 in radiobiology. A detailed description of the courses appears elsewhere in this issue of the JOURNAL.

SOCIETY PROCEEDINGS AND NEWS ITEMS

AMERICAN BOARD OF RADIOLOGY ELECTS OFFICERS

The following officers were elected at the meeting of the American Board of Radiology in Chicago, Illinois, May 19–23, 1958: Bernard P. Widmann, President; Laurence L. Robbins, Vice-President; H. Dabney Kerr, Secretary; and James W. J. Carpender, Treasurer.

THE AMERICAN BOARD OF RADIOLOGY

The American Board of Radiology announces the following examinations:

Regular examination, December 8–11, 1958, Washington, D. C.; final date for filing applications was July 1.

Special examination in Nuclear Medicine for those diplomates in Radiology or Therapeutic Radiology, December 6, 1958, Washington, D. C.; deadline for filing applications is October 1.

Special examination, March 16–19, 1959, Cincinnati, Ohio; deadline for filing applications is November 1, 1958.

Candidates completing training June 30, 1959 are not eligible for the Spring 1959 examinations.

The Board of Trustees of the American Board of Radiology has decided against giving a written examination.

SURGEON GENERAL'S OFFICE TO PUBLISH RADIOLOGICAL VOLUME

The Historical Unit of the Surgeon General's office of the United States Army Medical Corps is to publish a volume devoted entirely to radiology performed in World War II by physicians serving in the United States Army Medical Corps.

Heretofore, the activities of the Radiologist in the United States Army Medical Corps have been included in the volumes on surgery. It is welcome news to know that the work of those radiologists who performed in the United States Army Medical Corps will be memorialized in a volume exclusively devoted to their services.

Doctor Kenneth Allen requests that the Radiologists who have photographs, journals, diaries, letters, and such appropriate reminiscences which would be of great value to the editorial staff should be sent to him as Editor of the forthcoming volume dealing with radiology performed in World War II. Due credit for the material used will be given to the contributor, and such loan material will, of course, be returned to the person sending it in.

The publication will appear as a United States Surgeon General's Office publication through the Historical Unit, and will be prepared under the supervision of an editorial Board of Radiologists and Doctor Allen as Chairman of the Board.

Those who served in World War II having such information as above requested will please communicate with Doctor Allen, whose address is: Dr. Kenneth D. A. Allen, Editor, 800 E. 8th Avenue, Denver 18, Colorado.

COURSE IN CLINICAL USE OF RADIOACTIVE ISOTOPES

A course in the clinical use of radioactive isotopes will be given by I. F. Hummon, M.D. and Robert S. Landauer, Ph.D. and associates at the Cook County Hospital, September 29 through October 10, 1958, Monday through Friday, from 9:00 A.M. to 12:00 M. and 1:00 P.M. to 4:00 P.M.

This is a full-time course including lectures, demonstrations, laboratory periods, and clinics. Lecture subjects will include radiation physics, radiation hazards and protection, natural and artificial radioactivity, theory of measurement, and counters and statistics. Clinical subjects will include the preparation, administration and uptake of tracer materials, thyroid physiology, thyroid neoplasms, blood dyscrasias, blood volume studies, and other uses of iodine 131, phosphorus 32, and gold 198. Atomic Energy Commission requirements and the obtaining of radioactive isotopes will be discussed.

The course will be offered for a minimum of eight students and a maximum of fifteen students; the fee for the course is $200.00. Inquiries may be addressed to the Registrar, Cook County Graduate School of Medicine, 707 W. Wood Street, Chicago 12, Illinois.

SOCIEDAD MEXICANA DE RADIOLOGÍA

The Sociedad Mexicana de Radiología has acquired a building of its own, which is admirably adapted to its needs. It consists mainly of an auditorium, two fully equipped halls for the teaching of radiologic technique, both theoretical and practical, a library, lounge, cafeteria, rest rooms, et cetera, and last, but not least, a garden which makes for peace of mind and beauty. The new address of the Mexican Society of Radiology is Calle Coahuila 35, México 7, D.F. They will welcome the visit of any of the members of the American Roentgen Ray Society who are in Mexico City.

SIXTH INTER-AMERICAN CONGRESS OF RADIOLOGY

Dr. Vicente Ubillús, General Secretary, Sixth Inter-American Congress of Radiology, requests that those American Radiologists, who sent in their ap-

plication to the Sixth Inter-American Congress of Radiology and who did not receive the official acknowledgment of their membership, claim their membership card at the office of the Congress in Lima, Perú. The address of Dr. Vicente Ubillús is: Instituto de Radiología "Cayetano Heredia," Hospital Arzobispo Loayza, Av. Alfonso Ugarte, Lima, Perú.

AMERICAN ROENTGEN RAY SOCIETY

SECTION ON INSTRUCTION

HAROLD O. PETERSON, M.D., *Director*

Titles and Abstracts of Courses Offered

Fifty-ninth Annual Meeting
Shoreham Hotel, Washington, D. C.
September 30 to October 3, 1958

PRESIDENT-ELECT BARTON R. YOUNG, with the approval of the Executive Council, has requested that the Section on Instruction of the American Roentgen Ray Society be continued for the Annual Meeting of 1958. The courses will all be of ninety minutes' duration and will be held between the hours of 3:00 P.M. and 4:30 P.M. on Tuesday, Wednesday and Thursday and from 1:30 P.M. to 3:00 P.M. on Friday. No other official activity has been scheduled for this time.

Sixteen rooms are available for the Instruction Courses and one course will be offered in each room each afternoon of the Annual Meeting. A total of 57 instructors will give sixty-four courses during the four days.

There will be forty-four courses in Diagnostic Roentgenology and twenty courses in Therapeutic Radiology, Nuclear Medicine, Physics and Radiobiology. Of the diagnostic courses twenty-nine are single period courses; six are two period courses, and one is a three period course. In Therapy fourteen are single period courses and three are two period courses. It is desirable, although not absolutely essential, that one should sign up for both courses in case these appear to be sequential courses. This is particularly true of the courses given by Doctors Fletcher, Storaasli and Garcia.

In summary there are:

(1) Twenty-nine single period courses in Diagnostic Roentgenology; (2) six two period courses in Diagnostic Roentgenology; (3) one three period course in Diagnostic Roentgenology; (4) fourteen single period courses in Therapeutic Radiology; (5) three two period courses in Therapeutic Radiology.

These include one course in Chemotherapy; two in Nuclear Medicine; one in Physics; and two in Radiobiology.

GENERAL INFORMATION

Location of Courses

The majority of the courses will be held in rooms which are numbered 1009, 1010, 1011, 1012, 1013, 1014, 1015, 1016, 1017, 1018, 1019 and 1020. These rooms are located very near the Registration Desk. Four other rooms will also be used on the same general floor level as the Registration Desk, designed as the North Room, the South Room, and the Green Room, near the West elevator, and the Tamerlane Room to the right of the Lower Lobby. Full information will be available at the desk of the Section on Instruction, which will be located in the General Registration Area on Sunday afternoon, Monday afternoon and from 8:30 A.M. until 4:30 P.M. Tuesday, Wednesday, Thursday and Friday.

How to Register and Obtain Tickets for the Instruction Courses

Admission to the Instruction Courses will be by ticket only.

Following the list of titles and abstracts is a general order sheet. First, second, and third choices for each period should be selected carefully and indicated on the order

sheet. It is estimated that the number who can attend each course each day will range between 40 and 120 persons depending on room size. If the directions given on the order sheet are followed explicitly, errors in completing reservations for the courses will be minimized.

It is possible for one to attend only four periods of instruction, so the condensed schedule given on the last pages of this program should be consulted with care when registering and ordering tickets for the courses.

Reservations for the courses will be made in the order in which the order forms are received. Those who are not members of the American Roentgen Ray Society will be charged the nominal fee of $2.00 per period. Full-time graduate students in radiology will be admitted without fee, but they will be required to make application for the courses they wish to attend.

Previous to September 20, 1958, the order forms should be sent to the Director, Dr. Harold O. Peterson, 1995 West County Road "B," St. Paul 13, Minnesota. After September 20, 1958, the order forms should be sent directly to Dr. Harold O. Peterson, Shoreham Hotel, Washington, D. C.

To avoid the confusion always existing on the first days of the meeting, a set of tickets will be mailed to each person whose registration for courses reaches the office of the Director on or before September 20, 1958. The envelope containing the tickets will be labelled in a conspicuous manner. Registrants are urged to be on the lookout for their tickets and to bring them to the meeting. The success of this plan depends entirely on the cooperation of the registrants.

In case the courses are not filled by the time of the meeting, tickets will be available at the Registration Desk of the Section on Instruction on Sunday afternoon, September 28, and thereafter during the meeting.

Holders of Tickets

Those who do not have the proper ticket for the course they wish to attend will not be permitted to enter the room. An attendant will be at the door of each conference room to collect the tickets.

THE FACULTY

1. J. Robert Andrews, M.D., D.Sc. (Med.), Radiation Branch, National Cancer Institute, National Institutes of Health, Public Health Service, Department of Health, Education and Welfare, Bethesda, Maryland.
2. Robert P. Barden, M.D., Associate Professor of Radiology, Graduate School of Medicine, University of Pennsylvania and Radiologist, Chestnut Hill Hospital, Philadelphia, Pennsylvania.
3. Chauncey N. Borman, M.D., Assistant Clinical Professor of Radiology, St. Mary's Hospital, Minneapolis, Minnesota.
4. John Caffey, M.D., Professor of Radiology, College of Physicians and Surgeons, Columbia Presbyterian Medical Center, New York, New York.
5. John A. Campbell, M.D., Chairman, Department of Radiology, Indiana University Medical Center, Indianapolis, Indiana.
6. Giulio J. D'Angio, M.D., X-ray Therapy Department, The Children's Hospital, Boston, Massachusetts.
7. Juan A. del Regato, M.D., Director, Penrose Cancer Hospital, Colorado Springs, Colorado.
8. Bernard S. Epstein, M.D., Department of Radiology, The Long Island Jewish Hospital, New Hyde Park, Long Island, New York.
9. Lewis E. Etter, M.D., Director, Department of Radiology, The Falk Clinic; Consultant, Western Psychiatric Institute and Clinic; Professor of Radiology, School of Medicine, University of Pittsburgh, Pittsburgh, Pennsylvania.
10. Titus C. Evans, Ph.D., Head, Radiation Research Laboratory, College of Medicine, State University of Iowa, Iowa City, Iowa.
11. Benjamin Felson, M.D., Professor and Director, Department of Radiology, University of Cincinnati, Cincinnati, Ohio.
12. Gilbert H. Fletcher, M.D., Director of Radiation Therapy, M. D. Anderson Hospital and Tumor Institute, Houston, Texas.
13. Manuel Garcia, M.D., Director, Department of Therapeutic Radiology, Charity Hospital of Louisiana, New Orleans, Louisiana.
14. Robert A. Garrett, M.D., Professor of Urology, Department of Urology, Indiana University Medical Center, Indianapolis, Indiana.

15. Brinton B. Gay, Jr., M.D., Department of Radiology, Emory University, Atlanta, Georgia.

16. Alfred Gellhorn, M.D., Columbia University, College of Physicians and Surgeons, Institute of Cancer Research, New York, New York.

17. Roy R. Greening, M.D., Associate Professor of Radiology, University of Pennsylvania Hospital, Philadelphia, Pennsylvania.

18. Joseph Hanelin, M.D., Director, Department of Radiology, Newton-Wellesley Hospital, Newton, Massachusetts and Clinical Associate Radiologist, Massachusetts General Hospital, Boston, Massachusetts.

19. Philip J. Hodes, M.D., Professor of Radiology, University of Pennsylvania, Philadelphia, Pennsylvania.

20. Kenneth E. Hodge, M.B., Chief Radiologist, Department of Radiology, Sunnybrook Hospital, Toronto, Ontario, Canada.

21. Colin B. Holman, M.D., Section of Diagnostic Roentgenology, Mayo Clinic, Rochester, Minnesota.

22. John W. Hope, M.D., Director, Department of Radiology, The Children's Hospital of Philadelphia; Assistant Professor of Radiology, University of Pennsylvania, Philadelphia, Pennsylvania.

23. George Jacobson, M.D., Professor and Head of Radiology, University of Southern California, Los Angeles, California.

24. Thomas J. Kennedy, M.D., Denver, Colorado.

25. John Kirkpatrick, M.D., Department of Radiology, St. Christopher's Hospital, Philadelphia, Pennsylvania.

26. Edwin L. Lame, M.D., Director, Department of Radiology, The Presbyterian Hospital in Philadelphia, Philadelphia, Pennsylvania.

27. Richard G. Lester, M.D., Assistant Professor of Radiology, University of Minnesota, Minneapolis, Minnesota.

28. Emanuel J. Levin, M.D., Director of Radiology, Maimonides Hospital of Brooklyn and Professor of Radiology, State University of New York, Brooklyn, New York.

29. James E. Lofstrom, M.D., Chairman and Professor, Department of Radiology, Wayne State University, College of Medicine, Detroit, Michigan.

30. Alexander S. MacMillan, M.D., Director of Radiology, Massachusetts Eye and Ear Infirmary, Boston, Massachusetts.

31. Alexander R. Margulis, M.D., Assistant Professor of Radiology, University of Minnesota, Minneapolis, Minnesota; Captain, Radiology Service, U. S. Army Hospital, Fort Bragg, North Carolina.

32. Richard H. Marshak, M.D., Director of Department of Radiology, Mt. Sinai Hospital, New York, New York.

33. Charles L. Martin, M.D., The Martin X-ray & Radium Clinic, Dallas, Texas.

34. Donald L. McRae, M.D., Department of Neurology and Neurosurgery, McGill University, Montreal Neurological Institute, Montreal, Quebec, Canada.

35. Harry Z. Mellins, M.D., Professor and Chairman, Department of Radiology, State University of New York, Brooklyn, New York.

36. Jarrell E. Miller, M.D., Director of Radiology, Baylor University Hospital, Dallas, Texas.

37. Lowell S. Miller, M.D., Associate Radiotherapist, Department of Radiology, M. D. Anderson Hospital and Tumor Institute, Texas Medical Center, Houston, Texas.

38. Robert D. Moreton, M.D., Tom B. Bond Radiological Group, Fort Worth, Texas.

39. Donn G. Mosser, M.D., Associate Professor of Radiology, Director of Radiation Therapy, University of Minnesota, Minneapolis, Minnesota.

40. Walter T. Murphy, M.D., Director of Radiology, Roswell Park Memorial Institute, Buffalo, New York.

41. Charles M. Nice, Jr., M.D., Professor and Head of Radiology, Tulane University, New Orleans, Louisiana.

42. James J. Nickson, M.D., Chairman, Department of Radiation Therapy, Memorial Center for Cancer and Allied Diseases, New York, New York.

43. Francis F. Ruzicka, Jr., M.D., Director of Radiology, St. Vincent's Hospital of the City of New York; Clinical Professor of Radiology, New York University College of Medicine, New York, New York.

44. Richard Schatzki, M.D., Chief, Department of Radiology, Mount Auburn Hospital, Cambridge, Massachusetts; Clinical Associate in Radiology, Harvard Medical School, Boston, Massachusetts.

45. Robert Shapiro, M.D., Director, Department of Radiology, The Hospital of St. Raphael, New Haven, Connecticut.

46. Glenn E. Sheline, M.D., Department of Radiology, University of California Medical Center, San Francisco, California.

47. Frederic N. Silverman, M.D., Director, Division of Roentgenology, The Children's Hospital, Cincinnati, Ohio.

48. Norman Simon, M.D., Assistant Attending Radiotherapist, Mt. Sinai Hospital, New York, New York.

49. Herbert M. Stauffer, M.D., Professor and Director, Diagnostic Division, Department of Radiology, Temple University Hospital, Philadelphia, Pennsylvania.

50. John P. Storaasli, M.D., Department of Radiology, Western Reserve University, Cleveland, Ohio.

51. Juan M. Taveras, M.D., Department of Radiology, Neurological Institute, New York, New York.

52. William J. Tuddenham, M.D., Assistant Professor of Radiology, University of Pennsylvania, Philadelphia, Pennsylvania.

53. Eugene F. Van Epps, M.D., Professor and Director, Department of Radiology, University of Iowa, Iowa City, Iowa.

54. W. Walter Wasson, M.D., Denver, Colorado.

55. H. Stephen Weens, M.D., Emory University, School of Medicine, Atlanta, Georgia.

56. Marvin M. D. Williams, Ph.D., Section of Biophysics and Biophysical Research, Mayo Clinic, Rochester, Minnesota.

57. Robert E. Wise, M.D., Department of Radiology, The Lahey Clinic, and New England Baptist Hospital, Boston, Massachusetts.

DESCRIPTION OF COURSES

COURSE 101

Tuesday

JOHN CAFFEY, M.D.
New York, New York

1. Infantile Cortical Hyperostosis
2. Hypervitaminosis of Growing Bones
3. Trauma in Growing Bones

COURSE 102

Tuesday

ROBERT SHAPIRO, M.D.
New Haven, Connecticut

Metabolic Bone Diseases

Traditionally, much emphasis has been placed upon the role of the skeleton as a supporting structure. Considerably less attention has been focused on the homeostatic role of the skeleton in maintaining the composition of the body fluids. Because of its active participation in many important metabolic processes, the skeleton is subject to change in a number of systemic diseases. This course will attempt to present a logical classification of metabolic bone diseases and correlate the roentgen findings with the physiologic and chemical abnormalities.

COURSE 103

Tuesday

PHILIP J. HODES, M.D.
Philadelphia, Pennsylvania

Recent Advances in Body Section Radiography

There are many factors which bear upon effective and informative body section radiography. There are fundamentals without which the modality can prove to be a "hit or miss" proposition. In addition to the ordinary techniques, horizontal and erect positions, one is now aided immeasurably by the "book cassette," which makes it possible to save a great deal of time and radiation exposure without losing any information in the finished radiograph.

The fundamentals will be reviewed and considerable time will be spent in discussing the "book cassette." Demonstration of clinical applications of the modality will complete the session.

COURSE 104
and
COURSE 204

Tuesday and Wednesday

JOSEPH HANELIN, M.D.
Newton, Massachusetts

Cerebral Calcifications and Plain Film Findings of Brain Tumors

Course 104. Cerebral Calcifications. Gross calcium deposition may occur in a large group of intracranial lesions. The type and distribution of this calcification at times is quite distinctive, and at the very least should always evoke a carefully considered differential diagnosis. Conditions under discussion will include those of degenerative, vascular, infectious, traumatic and neoplastic etiology. Clinical correlation will be emphasized.

Course 204. Plain Film Findings of Brain Tumors.

Despite the free use of pneumography and angiography in the study of intracranial lesions, it is important to recognize and re-emphasize the value of the comparatively simple and innocuous plain film skull examination. This session will be devoted to a review of the pertinent changes in the calvarium and base of the skull as well as to displacement of physiologic calcification in the diagnosis of space-occupying masses. Certain normal variations which may simulate such abnormality will also be discussed.

COURSE 105
and
COURSE 205

Tuesday and Wednesday

JOHN W. HOPE, M.D.
Philadelphia, Pennsylvania

Techniques of Pediatric Radiology and Pediatric Radiologic Problems

Most departments of radiology are geared both physically and mentally to handle adults. That infants and children and the diseases and abnormalities with which they may be afflicted differ greatly from grown-ups is not generally appreciated. All too often a patient is referred to The Children's Hospital of Philadelphia because a roentgenogram has been said to show some serious abnormality. The parents of these children are terrified and often almost hysterical. With more careful technique and interpretation these serious abnormalities often resolve into perfectly normal findings. This course is designed to show some of these pitfalls, and to suggest how to avoid them.

Course 105 will deal with the chest, the neck, intravenous urography, and the skull.

Course 205 will deal with the abdomen and the gastrointestinal tract.

COURSE 106
and
COURSE 206

Tuesday and Wednesday

ALEXANDER S. MACMILLAN, M.D.
Boston, Massachusetts

Sinuses and Mastoids, Lateral Neck and Larynx

Course 106. Sinuses
Course 206. Mastoids, Lateral Neck and Larynx

COURSE 107

Tuesday

HERBERT M. STAUFFER, M.D.
Philadelphia, Pennsylvania
and
JOHN KIRKPATRICK, M.D.
Philadelphia, Pennsylvania

Image Intensification and Cinefluorography in Practice and Research

Experience with several types of image intensifiers will be described. Present applications, including use of closed-circuit television, in research and practice will be discussed and illustrated. Limitations of present devices will be considered. Since present instruments are particularly suited to pediatric practice, this aspect will receive special consideration.

COURSE 108
and
COURSE 208

Tuesday and Wednesday

GEORGE JACOBSON, M.D.
Los Angeles, California

Roentgen Diagnosis of Injuries of the Atlanto-Axial Joint

The atlanto-axial joint possesses unique features of structures and function which make it subject to certain rather distinctive injuries. Many of these can be demonstrated by rather simple examination, while others require more elaborate techniques.

The following will be discussed:

1. Normal anatomy and embryology
2. Normal motion of the atlanto-axial joint
3. Methods of examination
4. Readily recognizable fractures and dislocations will be dealt with briefly
5. The major portion of the period will be devoted to a discussion of injuries which are difficult to demonstrate and are somewhat unusual

Examination of the Heart by Conventional Radiology

Conventional radiography remains the basic diagnostic tool in the radiologic examination of the heart. Our understanding of both normal and abnormal cardiovascular anatomy and function has been greatly enhanced by cardiac catheterization and angiocardiography. As a result of the knowledge gained from these procedures, more precise interpretation of conventional radiographs has become possible. The following will be discussed:

1. Normal cardiac anatomy
2. Specific chamber enlargement
3. Estimation of pulmonary artery pressure and flow
4. Pulmonary changes associated with cardiac disease

Illustrative cases will be presented for analysis and discussion. The value of the posteroanterior roentgenogram of the chest will be stressed.

COURSE 109

Tuesday

FRANCIS F. RUZICKA, JR., M.D.
New York, New York

Portal Venography—Techniques, Interpretation and Clinical Application

Portal venography is a roentgen method which in a relatively short time has become useful in the study of abnormalities of the portal venous system and has earned an accepted position in the medical armamentarium of diagnostic procedures. It has practical application in the clinical problems of portal venous obstruction of both intrahepatic and extrahepatic types.

Cirrhosis (Laennec's cirrhosis in particular) is the most common cause of portal obstruction of the intrahepatic type. Portal hypertension with serious and frequently fatal gastrointestinal bleeding is one of the sequelae. The most effective means of therapy so far devised for the treatment of bleeding gastroesophageal varices is portacaval surgery. The portal venogram is of inestimable value to the surgeon in revealing immediate and detailed information concerning the anatomy and physiology of the portal venous system. Much operating time can thereby be saved by avoiding painstaking and difficult exploration, and indeed the venogram is of great aid in planning the operative procedure.

In the extrahepatic type of portal obstruction, the same is true. The site of venous block is readily demonstrated and the region of venous anatomy is defined. Location of a mass may be determined or confirmed. These and other practical applications of portal venography will be considered in this course.

Special attention will be paid to the technique of splenic portography performed percutaneously in the radiologic department, as well as to the portal portography performed at laparotomy in the operating room.

Normal and pathologic anatomy and physiology of the portal venous system will be reviewed to form a background for interpretation of the portal venogram. Features to be emphasized will include the site of venous block, collateral vein development, the streaming phenomenon, and other factors which influence roentgen visualization of the portal venous system.

COURSE 110

Tuesday

ALEXANDER R. MARGULIS, M.D.
Minneapolis, Minnesota

Arteriography of Extremities

The indications and value of arteriography of extremities will be discussed. Technique and contrast media will be considered in detail. The normal anatomy will be presented and features of various peripheral vascular diseases will be outlined. The importance of studying the small vessels as well as the large arteries will be stressed. Occlusive disease, arteriovenous communications, bone and soft tissue tumors will also be discussed. It will be emphasized that peripheral arteriography, due to progress in techniques, has become a relatively harmless procedure which can be done on an out-patient basis.

COURSE 111

Tuesday

HARRY Z. MELLINS, M.D.
Brooklyn, New York

Roentgen Diagnosis of Some Acute Abdominal Conditions

The course will present an analytic approach to the diagnosis of the acute inflammatory processes in the abdominal viscera, mesenteric arterial and venous thrombosis, trauma to the abdominal organs and the effects of some toxic agents, such as narcotics.

COURSE 201

Wednesday

ROBERT D. MORETON, M.D.
Fort Worth, Texas

Roentgen Diagnosis of Diseases of the Colon

The conduct of double contrast examinations of the large intestine will be discussed with emphasis on results with different barium mixtures, preparation of the patient, and technique of the examination. Some of the poor results will be shown and an attempt made to analyze the causes. Lastly, the advantages of double contrast examinations in both benign and malignant lesions involving the colon will be illustrated.

COURSE 202

Wednesday

COLIN B. HOLMAN, M.D.
Rochester, Minnesota

The Selection and Application of Roentgenologic Procedures in the Presence of Intracranial Bleeding

In the presence of known intracranial bleeding, the roentgenologist often shares with the neurosurgeon the responsibility of the hazard as well as of the evaluation of diagnostic procedures. This discussion will, therefore, concern itself with the choice and application of the various roentgenologic methods with regard to the safety and expected diagnostic yield, in addition to the evaluation of diagnostic criteria. Examples from actual cases will illustrate the individual lesions, their diagnosis, management, subsequent history and prognosis.

COURSE 203

Wednesday

W. WALTER WASSON, M.D.
Denver, Colorado
ROBERT P. BARDEN, M.D.
Philadelphia, Pennsylvania
ROY R. GREENING, M.D.
Philadelphia, Pennsylvania
THOMAS J. KENNEDY, M.D.
Denver, Colorado
RICHARD G. LESTER, M.D.
Minneapolis, Minnesota

Symposium on the Physio-Dynamics of Respiration, and the Lesser Circulation with Practical Applications

I. A brief historical sketch of the researches which have made this presentation possible.
II. The anatomy and the physiology of the lungs and the physio-dynamics which concern respiration, the lymphatic flow, and the lesser circulation.
III. Presentation of cases with the roentgen findings and discussion of the influences of the physio-dynamics and the alterations of the normal physiology of respiration, the lymphatic flow, and the lesser circulation, which has produced the roentgen findings.
  A. Cor pulmonale in emphysema and lung fibrosis
  B. Sudden death from lung embolus
  C. The diaphragmatic syndrome and obesity
  D. The closure of the ductus arteriosus
  E. Hypoventilation and atelectasis
  F. Mitral disease and lung congestion
  G. Kyphosis and heart failure
  H. Pulmonary edema and allergic responses

COURSE 207
COURSE 307
and
COURSE 407

Wednesday, Thursday and Friday

JAMES E. MILLER, M.D.
Dallas, Texas

Roentgen Examination of the Stomach and Small Bowel

The roentgen examination of the stomach and small bowel is important. Not all radiologists are able to maintain adequate skill in interpretation of special studies such as cerebral angiography, pneumography, angiocardiography and other similar procedures when they have little or no opportunity to see them. The upper gastrointestinal tract study, however, is one of the most frequently performed examinations where the radiologist plays a dominant role. It is necessary, therefore, that he acquire and maintain a superior skill in performing these examinations as well as interpreting the results.

Course 207 will deal principally with techniques of the upper gastrointestinal tract and small bowel examination. The minimum film requirement for an adequate upper gastrointestinal tract study will be demonstrated. Techniques for combination procedures which can be done without compromising any of the examinations will also be shown. Pitfalls and errors in interpretation will be discussed along with methods of avoiding them.

Course 307 will comprise the demonstration of the various lesions which can be diagnosed by means of this examination plus the unusual lesions which one encounters in a busy practice. If the group desires, the last half hour of the second period will be used for a quiz session.

Course 407 will be a quiz session with audience participation.

COURSE 209
and
COURSE 309

Wednesday and Thursday

LEWIS E. ETTER, M.D.
Pittsburgh, Pennsylvania

Normal and Pathologic Roentgen Anatomy of the Skull

Course 209. Description of standard techniques and positions with illustrative slides for roentgen examination of the skull and paranasal sinuses. Comparison of advantages of one projection over another with respect to visibility of particular structures. General considerations with reference to the cerebral and facial components of the cranium and different skull types. Analysis of individual bone components in producing the various lines

and shadows seen in plain roentgenograms of the skull and alteration of these structures by pathologic processes. These analyses will be applied to a study of the sphenoid, frontal, ethmoid, parietal and occipital bones in several projections.

Course 309. Detailed roentgen anatomic analyses of the paranasal sinuses and mastoids with description of roentgenographic techniques employed. Study of normal and pathologic alteration of roentgen features of the middle cranial fossa and their relation to the sphenoid sinuses. Consideration of the orbits and optic foramina with special attention to the frontal, ethmoid and sphenoid bone components in various projections as well as the features contributed by the zygomatic bones. In conclusion, the maxillofacial components of the cranium will be examined, showing some pathologic alterations, including the lacrimal canals and soft tissue delineation.

COURSE 210

Wednesday

RICHARD SCHATZKI, M.D.
Cambridge, Massachusetts

The Use of Gravity Maneuvers in the Examination of the Stomach

The use of gravity in the study of the stomach was forced upon the radiologist for the examination of massively bleeding patients. The inadvisability of palpation, far from being a handicap, proved to be a blessing in disguise, necessitating various maneuvers to examine every part of the stomach successfully.

These procedures have been found helpful for practical use in the examination of all stomachs. They have proved particularly useful in the study of the upper part of the stomach.

Systematic examination of the stomach with such gravity maneuvers will be described. Its practical value in the differential diagnosis of ulcer, cancer and other lesions of the stomach will be discussed.

COURSE 211
and
COURSE 311

Wednesday and Thursday

EMANUEL J. LEVIN, M.D.
Brooklyn, New York

Differential Diagnosis of Diseases of the Bones

There are characteristic radiologic patterns produced by diseases of the bones which permit a definite and accurate diagnosis. The reactions in the soft tissues, periosteum and cartilage are often of greater differential value than the changes in bone. Furthermore, many osseous diseases have peculiarities of destruction, production and localization which in themselves are of diagnostic significance. The detection and correct assessment of the varied roentgen patterns will permit an accurate diagnosis in a high percentage of cases.

An analytic approach to the differential diagnosis of diseases of the bones will be presented with emphasis placed on fundamental aspects.

I. Explanation and description of terms
   - A. Soft tissue reactions
   - B. Periosteal reactions
   - C. Osseous reactions

II. Tumors
   - A. Benign
   - B. Malignant

III. Infections
   - A. Acute
   - B. Chronic

COURSE 301

Thursday

ROBERT E. WISE, M.D.
Boston, Massachusetts

Intravenous Cholangiography

Intravenous cholangiography today is an accepted procedure and has proved to be of great value in the examination of the postcholecystectomy patient as well as those in whom the gallbladder has not been removed. Its greatest value lies in the examination of the patient who returns after cholecystectomy with the same or new symptoms. In certain instances, it has been of definite value in the precholecystectomy patient. Generally speaking, it should not be a substitute for routine oral cholecystography.

Technique and reactions will be considered in detail. Many pitfalls have been encountered and these will be presented. Since the procedure has been found to be most useful in the postcholecystectomy patient, the major portion of the time will be utilized in a discussion of the findings in this group of patients. Data related to the size and drainage of the common bile duct, obstruction (due to calculi, tumors, strictures, and fibrosis of sphincter of Oddi) have been analyzed and correlated with operative findings in a high percentage of the group. Emphasis will be placed upon the correlation of operative findings and the preoperative picture.

The procedure has been of considerable value in leading to definitive surgical treatment and subsequent relief in cases of partial obstructions of the biliary tract. Perhaps of even greater importance, however, has been its value in eliminating needless exploratory operations on the biliary tract.

COURSE 302

Thursday

DONALD L. MCRAE, M.D.
Montreal, Quebec, Canada

Intracranial Meningiomas (Meningeal Fibroblastomas)

The meningiomas make up almost 15 per cent of intracranial tumors. Since most of them are theoretically benign it is of great importance to recognize them before they damage or kill the patient, if at all possible.

The gross and microscopic features of these tumors will be discussed and demonstrated with colored photographs and micrographs. An attempt will be made to correlate the pathologic and radiologic findings by comparing the roentgenograms and the photographs made at operation. Postoperative roentgenographic control will be stressed. Radiation therapy, both preoperative and postoperative, will be considered.

COURSE 303

Thursday

KENNETH E. HODGE, M.B.
Toronto, Ontario, Canada

Placental Localization—Including Plain-Film Gravitational Methods and Pelvic Angiography

The various methods used for placental localization are reviewed. Particular attention is paid to the gravitational displacement technique, including details of interpretation and a statement of its practical value and accuracy in the diagnosis or exclusion of placenta previa.

A simple technique of pelvic angiography with simultaneous bilateral filling of the pelvic vessels by percutaneous femoral puncture is described together with interpretation of the films and an assessment of the applications in placental localization and other pelvic conditions.

COURSE 304

Thursday

EUGENE F. VAN EPPS, M.D.
Iowa City, Iowa

Diagnosis of Pneumonias in Infancy and Early Childhood

This course will present the correlation of the roentgenologic appearance of acute pneumonias, especially in infants, with the pathologic findings. An attempt will be made to clarify the errors made by radiologists in calling a chest roentgenogram with severe pneumonitis normal, because it looks so clear, and the error of calling a healthy chest abnormal.

COURSE 305

Thursday

CHARLES M. NICE, JR., M.D.
New Orleans, Louisiana

Roentgen Manifestations of Collagen Diseases in the Chest

A series of 109 patients with five collagen diseases admitted to the University of Minnesota Hospitals during the past fifteen years has been reviewed. Roentgenograms of the chest probably did not contribute to the diagnosis at any time in about one-third of the patients. In another one-third, one or two abnormalities appeared in the chest at some phase of the disease process, serving as confirmatory findings which could be considered of help in following the progress of the patient. Of special interest to the roentgenologist, however, is the final one-third of the patients in whom a combination or sequence of roentgen findings should actually lead one to suspect a collagen disorder. If there are clinical and laboratory findings suggesting the presence of a collagen disease, these roentgenographic changes will aid in the differentiation of the various types in about one-fifth of the patients.

There are many roentgen findings in the chest which appear in various collagen disorders, most of which are nonspecific. A nonspecific type of cardiac enlargement, interstitial pneumonitis and pleural effusion are signs which may be seen in any of the collagen diseases either individually or in combination. Combinations of these and other signs, such as hilar vascular enlargement, small nodules in the lungs, interstitial pulmonary fibrosis and other less commonly observed signs, may be of great help in actually diagnosing or following the progress of the condition or in evaluating the response to therapy.

COURSE 306
and
COURSE 406

Thursday and Friday

BENJAMIN FELSON, M.D.
Cincinnati, Ohio

Some Fundamentals in Chest Roentgen Interpretation

Chest roentgen interpretation should not merely represent an attempt to correlate particular shadows with specific diseases, but should be approached with an understanding of the basic principles of anatomy, physiology, pathology, and radiology involved. Some of these principles will be considered and their practical application illustrated.

Among the subjects to be discussed are:

Course 306. (1) A method of fluoroscopy; (2) the "silhouette" sign; and (3) the air bronchogram.

Course 406. (1) Anatomic variations in the pul-

monary fissures; (2) lobar collapse; (3) lobar enlargement; and (4) the disrupted fissure.

COURSE 308
and
COURSE 408

Thursday and Friday

JOHN A. CAMPBELL, M.D.
Indianapolis, Indiana
and
ROBERT A. GARRETT, M.D.
Indianapolis, Indiana

Radiology of the Genitourinary Tract

The roentgen signs of genitourinary tract disease will be presented from the standpoint of their technical dependence, specificity, limitations, and application to the differential diagnosis of various lesions. These lesions will be considered in pathologic groups which give rise to somewhat similar roentgen findings. An attempt will be made to show how concomitant processes may obscure or vary the roentgen picture, or produce indistinguishable findings. The illustrative case material of diagnositc problems will be supplemented with clinical data so that the possibilities of objective roentgen interpretation may be evaluated. The indications for special methods of roentgen investigation will be discussed as the specific instances present themselves during the course.

The course will be given in two periods according to the following outline.

Period I. Radiology of Genitourinary Tract
- A. Significant anomalies and normal variants
- B. Obstructive lesions
- C. Infections

Period II. Radiology of Genitourinary Tract
- A. Urolithiasis
- B. Retroperitoneal mass lesions
- C. Urinary tract trauma

COURSE 310

Thursday

BERNARD S. EPSTEIN, M.D.
New Hyde Park, Long Island, New York

The Radiologic Diagnosis of Tumors of the Mid-Brain and the Posterior Fossa

This course will consider the radiological diagnosis of tumors of the mid-brain and the posterior cranial fossa, including the cerebellum, the tentorium and high spinal cord tumors intruding into the foramen magnum. The clinical picture will be correlated with the plain films, pneumoencephalograms and subsequent observation at operation and postmortem examination. The application of cerebral angiography to these problems will be discussed briefly.

COURSE 401

Friday

JUAN M. TAVERAS, M.D.
New York, New York

Cerebral Angiography in Children

In the course the clinical and roentgenologic aspects of cerebral angiography in children will be discussed in detail. Emphasis will be placed on the normal anatomy and physiology of the cerebral circulation in children.

COURSE 402

Friday

RICHARD G. LESTER, M.D.
Minneapolis, Minnesota

Roentgen Diagnosis of Cardiac Lesions in Infants and Children

Since the advent of corrective surgical procedures for many of the cardiac lesions of childhood, the necessity for accurate diagnosis in these patients has become most important. In this course, the place of roentgengology in the clinical examination of patients with heart disease in the pediatric age range will be outlined. Interpretation in terms of pathologic anatomy and physiology will be stressed.

Correlation of the roentgen findings with the other clinical procedures and examinations will be presented, and the team approach to diagnosis of cardiac lesions emphasized. The use of conventional roentgen techniques, their value and limitations will be discussed at length. Special procedures, such as angiocardiography and retrograde aortography will be used for confirmation and further discussion, but this course will be oriented to the methods available to all radiologists.

COURSE 403

Friday

WILLIAM J. TUDDENHAM, M.D.
Philadelphia, Pennsylvania

New Concepts in Radiographic and Interpretative Techniques

The inadequacy of "density," "resolution," "contrast" and "unsharpness" as criteria for evaluating the "quality" of diagnostic roentgenograms will be discussed, and new criteria of "film quality" will be presented.

The significance of the various physical factors affecting the "quality" of roentgenograms will be considered, and the indications for so-called "high-kilovoltage" and "supervoltage" roentgenography as well as for the use of the "micro-focus" roentgen-ray tube will especially be noted.

Recent observations concerning view-box and sur-

rounding illumination, the control of glare, the choice of reading lenses—practical aids for the radiologist in perceiving critical "threshold" images—will be presented.

Pertinent experimental findings will be cited, but the practical clinical application of these concepts will be emphasized through the presentation of illustrative diagnostic roentgenograms.

COURSE 404

Friday

CHAUNCEY N. BORMAN, M.D.
Minneapolis, Minnesota

Bronchopulmonary Segmental Anatomy and Bronchography

Segmental pulmonary resections make necessary an exact determination and an accurate description of the location and extent of pulmonary disease. Segmental anatomy of the prevailing pattern of the bronchial tree with the common variations significant to the roentgenologist will be reviewed. Terminology in description of bronchial anatomy will also be discussed. Inflammatory disease patterns and bronchial obstructing lesions will be demonstrated. Technique of bronchography as a team procedure will be briefly outlined. The value and importance of complete bronchial mapping, permitting identification of all ten segments and their subsegments will be stressed.

COURSE 405

Friday

EDWIN L. LAME, M.D.
Philadelphia, Pennsylvania

Oral Cholecystography: Some Recent Lessons in Interpretation

In recent years an attempt has been made to improve the accuracy and value of oral cholecystography.

If it remains a perfunctory test for technicians only, it will provide rather little help. A method is described wherein the *clinical* radiologist does the thinking and most of the work, so that the test is really something of a consultation and not a mere "snapping of pictures" by a robot.

A short description will be given of a method considered adequate and contemporary: a concise and specific history-taking by the radiologist; fluoroscopy of the chest and abdomen with on-the-spot roentgenographic exposures; supplementary roentgenographic study in other special postures; reexamination with different or additional opaque medium; personal discussion with the clinician after the test is finished; and a new and more informative method of roentgenologic reporting.

Some classic errors regarding concentration of the opaque medium and fat contraction will be demonstrated.

The use of this test in illuminating lesions other than those of the biliary tract will be described: diseases of the esophagus, stomach, duodenum and liver which may account for false positive cholecystograms.

Intravenous study, so thoroughly discussed in the recent literature, will not be part of this course.

COURSE 409

Friday

FREDERIC N. SILVERMAN, M.D.
Cincinnati, Ohio

Roentgenologic Examination of the Genitourinary Tracts in Childhood

Abnormalities of the genitourinary tracts in childhood are commonly of importance because of obstruction to urinary flow. Mass lesions, obstructive or not, are of immediate concern because of the frequency of genitourinary sites of origin for intra-abdominal neoplasms. In addition, congenital malformations, which are far from uncommon, may present as obstructive lesions and/or mass lesions. Remedial surgery has become increasingly available, and requires, in many instances, only the accurate delineation of the anatomic and functional disturbances. This can be provided by present day techniques of roentgenologic examination to a degree not obtained by other techniques of examination.

This course will review, with the aid of slides, obstruction, mass and congenital lesions of the genitourinary apparatus of practical importance. Techniques will be discussed only insofar as special variations are required to demonstrate special conditions; a knowledge of standard techniques in normal anatomy will be assumed.

COURSE 410

Friday

RICHARD H. MARSHAK, M.D.
New York, New York

The Small Intestine

1. Techniques; including a discussion of the various barium preparations
2. Concepts of small intestinal patterns
3. The malabsorption syndrome. Sprue, lymphosarcoma and Whipple's disease
4. Regional enteritis
5. Infarction of the small bowel
6. Tumors

COURSE 411

Friday

H. STEPHEN WEENS, M.D.
Atlanta, Georgia
and
BRINTON B. GAY, JR., M.D.
Atlanta, Georgia

The Radiology of the Pulmonary Circulation in Health and Disease

Normal Anatomy: Pulmonary arteries and veins; pulmonary lymphatics; bronchial arteries

Physiology: Normal pressure values in pulmonary arteries, capillaries and veins; normal pulmonary blood flow

Special Roentgenologic Techniques: Fluoroscopy with image amplifier; laminagraphy; angiocardiography

Congenital Abnormalities: Agenesis of pulmonary artery; hypoplasia of pulmonary artery; abnormal pulmonary arteries and pulmonary sequestration; arteriovenous fistula

Abnormal pulmonary venous return

Acquired Diseases of Pulmonary Circulation: Pulmonary artery embolism and thrombosis; pulmonary infarction

Pulmonary artery dilatation and aneurysm

Pulmonary congestion and edema; Kerley's lines

Hemosiderosis and osteosis in mitral valvular disease

Hydrothorax

Physiologic Changes of Pulmonary Circulation: Increased pulmonary blood flow; decreased pulmonary blood flow; pulmonary hypertension

Correlation of pulmonary artery size and pulsation with pulmonary blood flow and pressure

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COURSE 121
and
COURSE 221

Tuesday and Wednesday

GILBERT H. FLETCHER, M.D.
Houston, Texas

Clinical Experiences with 2,000 Cases Treated by Supervoltage and Megavoltage Therapy

Carcinomas of the (1) uterine cervix, (2) head and neck, (3) urinary bladder, and (4) breast have been the main groups of cancers treated by supervoltage therapy. Another group are those tumors (testis, ovary, lymphomas), necessitating large volume torso therapy.

Details of techniques and trends in results will be given for each of these groups.

COURSE 122
and
COURSE 222

Tuesday and Wednesday

JOHN P. STORAASLI, M.D.
Cleveland, Ohio

Radioactive Isotopes—Therapeutic Application

A review will be made of the more important therapeutic applications of radioisotopes. Included will be a discussion of the rationale of therapy and a comparison of other methods of treatment. The isotopes to be covered in detail will be radioactive phosphorus in the treatment of polycythemia, leukemia, and metastatic breast carcinoma in bone; radioactive iodine for diseases of the thyroid gland and euthyroid cardiacs; radioactive colloidal gold for malignant serous effusions; and radioactive strontium for beta ray therapy of ocular lesions.

COURSE 123

Tuesday

TITUS C. EVANS, Ph.D.
Iowa City, Iowa

Radiation Biology

Current concepts and recent literature concerning biologic effects of ionizing radiation will be discussed. Topics such as mechanism of action, effects of protraction, fractionation, linear energy transfer, modifying agents, etc., will be examined with a view of applying them as much as possible to radiation therapy and radiation protection problems.

COURSE 124

Tuesday

DONN G. MOSSER, M.D.
Minneapolis, Minnesota

Through Twentieth Century Europe with Roentgen and Leica

Visits to European radiotherapeutic centers have become a popular and valuable experience for radiologists. Potential radiologic itinerants have frequently solicited advice prior to such travels.

This course is designed to fulfill the needs for information of those who are planning trips to Europe and those interested in hearing one point of view on European radiation therapy. It is based on the experience of the instructor, his wife, and their three grade school age children during thirteen months in 1954 and 1955 on an American Cancer Society Fellowship in numerous clinics in Great Britain, Scandinavia, France, Germany and Switzerland.

Kodachrome slides will be used to illustrate suggestions as to whom to see, when to go, how to get what you want, and how to cope with some of the

problems associated with a medical odyssey of this type.

Particular attention will be given to the Manchester radium techniques, the McWhirter technique for breast carcinoma, the Stockholm techniques for gynecologic lesions, and other European radiation therapy procedures that are likely to be of interest.

Moving field therapeutic applications in various clinics will be illustrated. Details of dose planning techniques in various clinics will be described. Differences between various European and American approaches will be discussed.

COURSE 125
and
COURSE 225

Tuesday and Wednesday

MANUEL GARCIA, M.D.
New Orleans, Louisiana

The Treatment of Carcinoma of the Cervix

This course on the treatment of carcinoma of the cervix by radiation will cover two periods. In the first period, the nosographic background essential for the conduct of treatment will be considered and the physical principles of recommended radium and roentgen-ray techniques will be presented. The second period will deal with the biophysical factors in dosimetry, tolerance and tumor dosage, and the modifications in treatment planning resulting from characteristics of the host, effects of the tumor on the patient, and features of the neoplasm. Finally, the reactions, injuries, and the results of treatment will be considered.

COURSE 223

Wednesday

JAMES J. NICKSON, M.D.
New York, New York

Palliative Management of Distant Manifestations of Carcinoma

The purpose of this course is to discuss some of the more common sites for metastases, the indications and the principles underlying radiologic management and the results of such management. The indications and contraindications for the use of combined simultaneous chemotherapy or hormonal medication with roentgen treatment will be considered.

The material will be organized according to the organ or tissue involved.

1. *Osseous metastases*

The cancers most frequently producing osseous metastases arise from the breast, lung, kidney and prostate.

Indications for treatment are usually pain and threatened or actual pathologic fracture. The probability of relief of pain in osteolytic metastases is approximately 75 per cent.

Impending fracture can only maximally be helped if two things occur: decrease in activity of the neoplastic cell and increase in bone salt deposition.

We commonly give 2,000 r tissue dose in two weeks with 250 kv. radiation to the osteolytic or mixed lesion.

Osteoblastic lesions are ordinarily treated with 2,500 to 3,000 r in three weeks. Limited experience would indicate that probability for relief of pain is not as great as with osteolytic lesions.

2. *Intracranial metastases* most frequently occur from lung and breast cancer.

Treatment, since these lesions are usually multiple, is directed to the entire intracranial contents with 3,000 r being delivered in three weeks.

3. *Spinal cord compression* may or may not be associated with obvious osseous disease. It is most commonly seen in lymphomas and in carcinoma of the breast. Care must be taken, however, to establish the correct level of compression. Since permanent damage to the cord will occur if the compressing lesion is not rapidly reduced in size, treatment should be begun as soon as the diagnosis is established. This constitutes one of the two radiotherapeutic emergencies.

4. *Liver metastases* occur most commonly from carcinoma of the breast and carcinoma of the lung. Indications for treatment and results will be presented briefly.

5. *Intrapulmonary metastases* are seen most frequently from carcinoma of the large bowel, carcinoma of the breast and renal carcinoma. Treatment should be, in general, reserved for the lesions producing symptoms or dyspnea or those which in the judgment of the responsible physician will shortly do so.

COURSE 224

Wednesday

JAMES E. LOFSTROM, M.D.
Detroit, Michigan

Cobalt 60 Brachytherapy Devices: Clinical and Biophysical Considerations

During the past few years there have become available new forms of cobalt 60 devices such as beads, disks, plaques and cups with broad potentials of clinical value. They offer a usage which may be superior to radium in a great many instances. Proper application is dependent upon an adequate knowledge of not only the physical aspects of this radiation source, but also certain radiobiologic effects. These will be set forth, based on comparative experi-

mental studies using 250 kv. and cobalt 60. Representative isodose patterns for single sources and combinations of multiple sources will be demonstrated and their application in several clinical situations will be discussed such as in the instance of neoplasms of the skin, endometrium, antrum, and bladder.

COURSE 321

Thursday

ALFRED GELLHORN. M.D.
New York, New York

Chemotherapy in Disseminated Neoplastic Disease (Including Lymphomas)

The material to be covered includes a discussion of the indications, contraindications, and toxicity of drugs in the treatment of disseminated neoplastic disease. Emphasis will be placed on the use of hormones, antimetabolites and alkylating drugs. The relationship of chemotherapy and radiation therapy in the management of human cancer and lymphomas will be stressed.

COURSE 322

Thursday

CHARLES L. MARTIN, M.D.
Dallas, Texas

The Treatment of Cancer of the Head and Neck with Irradiation

This course deals with the treatment of cancer of the face, nose, eye, ear, lip, intraoral cavity and oropharynx and of the metastatic cervical lymph nodes with irradiation alone. The techniques which will be considered utilize roentgen rays generated at 85 and 220 kv., and implanted low intensity radium needles. Many lantern slides will be shown illustrating the results obtained in individual patients and statistical compilations will also be given. Emphasis will be placed on the results obtained in cancer of the floor of the mouth, gums and metastatic cervical lymph nodes.

COURSE 323

Thursday

J. ROBERT ANDREWS, M.D., D.Sc. (Med.)
Bethesda, Maryland

Modification of the Radiation Response

Great interest has been aroused in the possibility of modifying the clinical radiation therapy response by introducing simultaneous combined treatment with cytotoxic drugs. Despite a substantial amount of clinical activity along these lines, there is, to date, no absolutely convincing evidence that either synergistic, or its opposite, antienergistic effects have been produced by the auxiliary use of any physical, chemical, or biologic agent in combination with ionizing radiations. The reasons for this and some possible avenues of approach to the solution of the problem will be discussed from the points of view of the basic mechanisms of the radiation response, the potentiality for step-wise modification of them by other agents, pharmacologic considerations of certain agents, and considerations of the empirical approach to clinical investigation, experimental design, and statistical evaluation.

The course is designed for practicing radiologists as an aid to an understanding and evaluation of the controversial literature on the subject.

COURSE 324

Thursday

GLENN E. SHELINE, M.D.
San Francisco, California

Radioiodine in Thyroid Disease

This course will include discussion of both diagnostic and therapeutic uses of radioiodine in thyroid disease; however, more emphasis will probably be placed on the therapeutic aspects. The thyroid uptake of I^{131} in various thyroid diseases will be considered and examples of scanning for nodules and aberrant thyroid tissue will be shown. An evaluation of the place of radioiodine in the treatment of thyrotoxicosis and thyroid carcinoma will be presented. The discussion will include indications, limitations and results of radioiodine therapy for these diseases.

COURSE 325

Thursday

NORMAN SIMON, M.D.
New York, New York

Cobalt 60 Therapy: Treatment Technique with a Hectocurie Unit

Cobalt 60 hectocurie teletherapy promises to replace 250 kv. roentgen therapy as "conventional" radiation therapy. Changes in technique of treatment are required because of the characteristics of cobalt 60 radiation, *i.e.*, sparing of skin, relatively low bone absorption and decreased side-scatter. The effect of these characteristics on treatment plans for cancers of the head and neck, breast, lung, esophagus, uterus and rectum will be discussed. The value of short source-skin distance in the treatment of accessible cancer is to be stressed.

The limitations of cobalt 60 hectocurie teletherapy include the problems of subcutaneous fibrosis, penumbra, low output, small field size and protection.

COURSE 421

Friday

GIULIO J. D'ANGIO, M.D.
Boston, Massachusetts

Radiation Therapy in the Management of Neoplasms of Infancy and Childhood

1. Review of patients (by diagnosis) seen in the Children's Medical Center X-ray Therapy Department
2. Discussion of the peculiarities of childhood tumors
3. Treatment programs for most common neoplasms of this age group. A brief general survey of apparatus and techniques will be included
4. The role of antitumor chemotherapeutic agents
   a. Present
   b. A look to the future
5. Late effects of radiation in infants and children
6. Survival rates

COURSE 422

Friday

JUAN A. del REGATO, M.D.
Colorado Springs, Colorado

Radiobiology for Physicians

In the numerous aspects of radiobiologic research in many directions in which it is justifiable to investigate, the physician finds himself often at a loss to seek the basic radiophysiologic information that is important to a clinical practice of radiation therapy. This course will attempt to present a summary of the important facts of radiobiology in clinical practice.

COURSE 423

Friday

LOWELL S. MILLER, M.D.
Houston, Texas

Treatment of Breast Carcinoma

Institutional policies in the management of breast carcinoma at the University of Texas M. D. Anderson Hospital and Tumor Institute, Houston, Texas—surgery alone for early cases, radiation therapy alone for late, and various combinations of the two for intermediate stages—will be discussed. Radiation therapy techniques will be detailed and illustrative cases presented.

COURSE 424

Friday

WALTER T. MURPHY, M.D.
Buffalo, New York

Roentgen Therapy of Skin Cancer

This course will illustrate clinical results of various techniques currently used at the Roswell Park Memorial Institute for small and large lesions involving various anatomic sites, with and without invasion of cartilage and bone. The discussion will accent (1) the advantages of small increment fractionation as compared to the so-called "massive-dose" technique, (2) the proper selection of radiation quality (half-value layer 0.06 mm. Al, 30 kv. peak up to half-value layer 0.9 mm Cu, 200 kv. peak) and the indications for combining different radiation qualities, (3) the influence of the cancer site upon the selection of field size, (4) the care of the patient during and following irradiation, and (5) the factors influencing complications and sequelae.

COURSE 425

Friday

MARVIN M. D. WILLIAMS, Ph.D.
Rochester, Minnesota

Tissue Dose: The Result of Transfer of Energy from Radiation to Tissue

The dose which tissue receives as a result of irradiation is the energy which the tissue absorbs. Because the transfer of energy from radiation to tissue begins with an interaction between radiation and atoms which produces energetic corpuscles, the nature of radiation and the structure of atoms will be reviewed. The various ways in which radiation interacts with atoms to produce corpuscles will be described. Next will follow a discussion of (1) the characteristics of the corpuscles and how these characteristics depend on the quality of the radiation which produced them, (2) the way in which the energy of the corpuscles is transferred to tissue and how this transference depends on the energy of the corpuscles, and (3) the relationship between the production of corpuscles within a small volume of tissue and the dose (energy absorbed) in that small volume.

CONDENSED SCHEDULE OF COURSES

TUESDAY **3:00 to 4:30 P.M.** **September 30, 1958**

Diagnostic Roentgenology		Therapeutic Radiology, Nuclear Medicine, Physics and Radiobiology	
101—Caffey	1. Infantile Cortical Hyperostosis 2. Hypervitaminosis of Growing Bones 3. Trauma in Growing Bones	**121—Fletcher**	Clinical Experiences with 2,000 Cases Treated by Supervoltage and Megavoltage Therapy
102—Shapiro	Metabolic Bone Diseases		
103—Hodes	Recent Advances in Body Section Radiography	**122—Storaasli**	Radioactive Isotopes—Therapeutic Application
104—Hanelin	Cerebral Calcifications		
105—Hope	Techniques of Pediatric Radiology and Pediatric Radiologic Problems		
106—MacMillan	Sinuses	**123—Evans**	Radiation Biology
107—Stauffer Kirkpatrick	Image Intensification and Cinefluorography in Practice and Research		
108—Jacobson	Roentgen Diagnosis of Injuries of the Atlanto-Axial Joint	**124—Mosser**	Through Twentieth Century Europe with Roentgen and Leica
109—Ruzicka	Portal Venography—Techniques, Interpretation and Clinical Application		
110—Margulis	Arteriography of Extremities	**125—Garcia**	The Treatment of Carcinoma of the Cervix
111—Mellins	Roentgen Diagnosis of Some Acute Abdominal Conditions		

CONDENSED SCHEDULE OF COURSES

WEDNESDAY **3:00 to 4:30 P.M.** **October 1, 1958**

Diagnostic Roentgenology		Therapeutic Radiology, Nuclear Medicine, Physics and Radiobiology	
201—Moreton	Roentgen Diagnosis of Diseases of the Colon	**221—Fletcher**	Clinical Experiences with 2,000 Cases Treated by Supervoltage and Megavoltage Therapy
202—Holman	The Selection and Application of Roentgenologic Procedures in the Presence of Intracranial Bleeding		
203—Wasson Barden Greening Kennedy Lester	Symposium on the Physio-Dynamics of Respiration, and the Lesser Circulation with Practical Applications	**222—Storaasli**	Radioactive Isotopes—Therapeutic Application
204—Hanelin	Plain Film Findings of Brain Tumors		
205—Hope	Techniques of Pediatric Radiology and Pediatric Radiologic Problems	**223—Nickson**	Palliative Management of Distant Manifestations of Carcinoma
206—MacMillan	Mastoids, Lateral Neck and Larynx		
207—Miller	Roentgen Examination of the Stomach and Small Bowel	**224—Lofstrom**	Cobalt 60 Brachytherapy Devices, Clinical and Biophysical Considerations
208—Jacobson	Examination of the Heart by Conventional Radiology		
209—Etter	Normal and Pathologic Roentgen Anatomy of the Skull		
210—Schatzki	The Use of Gravity Maneuvers in the Examination of the Stomach	**225—Garcia**	The Treatment of Carcinoma of the Cervix
211—Levin	Differential Diagnosis of Diseases of the Bones		

CONDENSED SCHEDULE OF COURSES

THURSDAY **3:00 to 4:30 P.M.** **October 2, 1958**

Diagnostic Roentgenology		Therapeutic Radiology, Nuclear Medicine, Physics and Radiobiology	
301—Wise	Intravenous Cholangiography	**321—Gellhorn**	Chemotherapy in Disseminated Neoplastic Disease (Including Lymphomas)
302—McRae	Intracranial Meningiomas (Meningeal Fibroblastomas)		
303—Hodge	Placental Localization—Including Plain-Film Gravitational Methods and Pelvic Angiography	**322—Martin**	The Treatment of Cancer of the Head and Neck with Irradiation
304—Van Epps	Diagnosis of Pneumonias in Infancy and Early Childhood		
305—Nice	Roentgen Manifestations of Collagen Diseases in the Chest	**323—Andrews**	Modification of the Radiation Response
306—Felson	Some Fundamentals in Chest Roentgen Interpretation		
307—Miller	Roentgen Examination of the Stomach and Small Bowel	**324—Sheline**	Radioiodine in Thyroid Disease
308—Campbell Garrett	Radiology of the Genitourinary Tract		
309—Etter	Normal and Pathologic Roentgen Anatomy of the Skull		
310—Epstein	The Radiologic Diagnosis of Tumors of the Mid-Brain and the Posterior Fossa	**325—Simon**	Cobalt 60 Teletherapy: Treatment Technique with a Hectocurie Unit
311—Levin	Differential Diagnosis of Diseases of the Bones		

CONDENSED SCHEDULE OF COURSES

FRIDAY **1:30 to 3:00 P.M.** **October 3, 1958**

Diagnostic Roentgenology		Therapeutic Radiology, Nuclear Medicine, Physics and Radiobiology	
401—Taveras	Cerebral Angiography in Children	**421—D'Angio**	Radiation Therapy in the Management of Neoplasms of Infancy and Childhood
402—Lester	Roentgen Diagnosis of Cardiac Lesions in Infants and Children	**422—del Regato**	Radiobiology for Physicians
403—Tuddenham	New Concepts in Radiographic and Interpretative Techniques		
404—Borman	Bronchopulmonary Segmental Anatomy and Bronchography		
405—Lame	Oral Cholecystography: Some Recent Lessons in Interpretation	**423—Miller**	Treatment of Breast Carcinoma
406—Felson	Some Fundamentals in Chest Roentgen Interpretation		
407—Miller	Roentgen Examination of the Stomach and Small Bowel	**424—Murphy**	Roentgen Therapy of Skin Cancer
408—Campbell Garrett	Radiology of the Genitourinary Tract		
409—Silverman	Roentgenologic Examination of the Genitourinary Tracts in Childhood	**425—Williams**	Tissue Dose: The Result of Transfer of Energy from Radiation to Tissue
410—Marshak	The Small Intestine		
411—Weens Gay	The Radiology of the Pulmonary Circulation in Health and Disease		

BOOK REVIEWS

Books sent for review are acknowledged under: Books Received. This must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interest of our readers as space permits.

MALADIES DES VEINES; DIAGNOSTIC ET TRAITEMENT. By Claude Olivier, Agrégé de la Faculté de Médecine de Paris, Chirurgien des Hôpitaux. Cloth. Price, 7,400 fr. Pp. 600, with 342 illustrations. Masson et C^ie^, Éditeurs, 120 boulevard Saint-Germain, Paris 6^e^, France, 1957.

This large monograph is based on 2,500 clinical cases, 1,500 phlebographic studies and several hundred patients treated by surgical, medical and various supportive measures. Therefore, it contains much of value for the roentgenologist, surgeon and other physicians dealing with diseases of veins.

The book is divided into nine large sections on the diagnosis and treatment of various types of venous disorders in various parts of the body. The ninth section is a special discussion of 58 pages on phlebography. Extensive lists of periodical references are given after the subdivisions and chapters. A bibliography of 118 monographs and supplements is given at the end of the text.

Each chapter is illustrated with superb roentgenographic reproductions. This is probably one of the most comprehensive individual works available in this field.

The monograph is enthusiastically recommended.

CHARLES M. NICE, JR., M.D.

DIE VASKULÄREN ERKRANKUNGEN IM GEBIET DER ARTERIA VERTEBRALIS UND ARTERIA BASIALIS; EINE ANATOMISCHE UND PATHOLOGISCHE, UND NEURORADIOLOGISCHE STUDIE. By Prof. Dr. H. Krayenbühl, Direktor der Neurochirurgischen Universitätsklinik Zürich; and Dr. G. Yaşargil, Assistent der Neurochirurgischen Universitätsklinik Zürich. Cloth. Price, DM 77.—. Pp. 170, with 125 illustrations. Georg Thieme, Herdweg 63, Stuttgart, Germany, 1957.

The embryology and anatomy of the vertebral and basilar arteries are discussed in great detail. The symptomatology of thrombosis of the vessels in the subtentorial region is approached from historical and personal studies. The authors believe that, because of the variation in blood supply of the medulla oblongata, it is impossible to make a differential diagnosis between a thrombosis of the vertebral artery or one of its branches. The rich blood supply and numerous anastomoses between the various vessels assure a sufficient blood supply to the vital centers of the subtentorial area.

Occasionally, an angiogram of the vertebral artery will show only the inferior posterior cerebellar artery. Without pathologic-anatomic confirmation, one should not conclude from this that the two vertebral arteries do not join together. The arterial pressure of the other vertebral artery may be sufficiently great to prevent the contrast material from entering the point of junction and into the basilar artery. The authors have visualized the contralateral vertebral artery in 7 per cent of their cases (270 angiograms). The anterior and posterior spinal arteries are visualized very seldom. In 88 per cent of cases, the inferior posterior cerebellar artery is demonstrated. The superior cerebellar arteries are visualized constantly. The posterior cerebral arteries lie as mirror images to one another in the anterioposterior view and near one another in the lateral view. This artery is not visualized in about 10 per cent of vertebral angiograms, but one should have an anterioposterior view to verify its absence. To make certain that there is or is not a thrombosis of this vessel, a carotid angiogram should also be done.

In carotid angiography, the posterior cerebral artery is visualized in 25–30 per cent of cases, due probably to a somewhat enlarged posterior communicating artery. The internal carotid artery and its branches are demonstrated in 1 per cent of vertebral angiograms. In thrombosis of the internal carotid artery, blood supply may be furnished by the vertebral artery through the posterior communicating vessel.

The sites of predilection for arteriosclerotic changes in the blood vessels of the brain are the internal carotid and basilar arteries. Thrombosis of the vertebral, basilar, and posterior cerebral arteries may be diagnosed on the angiograms, provided certain conditions are consid-

ered, such as spasm of a blood vessel, poor position of injection needle, marked retroflexion of the head, etc. The site of a thrombus in the vertebral artery may suggest surgical intervention. The authors believe that there are many patients with few or no symptoms following thrombosis of a vertebral artery, because of a sufficient compensation of blood supply by the contralateral vessel.

It is stated that 15–20 per cent of all intracranial aneurysms lie in the posterior portion of the circle of Willis, with one-half being in the basilar artery. The differential clinical diagnosis between a neoplasm and an aneurysm often is not possible.

There is a detailed discussion of cerebral vascular anomalies and neoplasms. Angiographic diagnosis of a cavernous angioma usually can be made, since in the arterial phase there is a marked displacement of the cerebral vessels and its branches, which finding very seldom is noted in the racemose arteriovenous angioma. Furthermore, there is a fine vascular ball which is situated only in one corner of the space-occupying process, suggesting a subcortical arteriovenous angioma or a glioblastoma. The youth of the patient is against a glioblastoma. For a definite diagnosis, localization, and anatomic structure of an arteriovenous aneurysm with its afferent and efferent vessels, bilateral carotid and vertebral angiograms are essential.

In conclusion, it is emphasized that the blood vessels in the subtentorial portion of the brain bear a constant and orderly relationship. The great difficulty in evaluating the vertebral angiogram is due, not to the great variations of the vessels, but to the fact that the vertebral artery is more difficult to puncture and one cannot easily obtain perfect angiograms. A poorly outlined vertebral angiogram should be considered a technical error and not abnormal. Thrombosis of the individual branches of the vertebral and basilar arteries, except for the posterior cerebral artery, cannot be diagnosed on the angiogram because these vessels may be absent normally. On the other hand, arteriosclerotic changes and thrombosis of the vertebral and basilar arteries can be diagnosed angiographically with certainty. Hemangioblastoma, although diagnosed clinically without difficulty, should be studied angiographically to determine if the tumor is cystic or rich in blood supply. Vertebral angiography has been of no value in subtentorial and subdural hematomas, and in intracerebellar and intrapontine bleeding.

The book is well organized and beautifully illustrated with sketches, photographs of specimens, and reproductions of angiograms. An extensive bibliography is added. The exhaustive study of the world literature is highly noteworthy. This book should be in the library of every neurologist, neuro-surgeon, and radiologist who deals with angiograms of the brain.

Samuel Richman, M.D.

DeMotu Cordis. By William Harvey; Translated from the original Latin by Kenneth J. Franklin, and now published for the Royal College of Physicians of London. Cloth. Price, $3.50. Pp. 209. Charles C Thomas, Publisher, 301 East Lawrence Avenue, Springfield, Illinois, 1957.

This attractive little volume commemorating a great event is a retranslation from the original Latin that was undertaken to present Harvey's first great report as accurately as possible. The result is a most lucid and moving account that erases time and places the reader in the midst of Harvey's rapt audience.

William S. Reveno, M.D.

Lehrbuch der röntgenologischen Differentialdiagnostik. Volume I. Erkrankungen der Brustorgane. By Prof. Dr. W. Teschendorf, Köln. Fourth edition. Cloth. Price, DM 210.—. Pp. 1,183, with 1,138 illustrations. Georg Thieme, Herdweg 63, Stuttgart, Germany, 1958.

This is the fourth edition of a text which was first issued in 1939, in 803 pages and with 815 illustrations. Its then relatively novel approach consisted in the discussion of the various diagnostic possibilities expected in the presence of a given morphologic alteration. Thus part (A) —pathology in the lungs proper—begins with the "opaque hemithorax" (associated with increase or with decrease in size), and continues with the descriptive changes, such as triangular, nodular, linear shadows encountered in chest roentgenograms. The intention is to teach the reader how to orient his thinking, and also to provide a source of reference, where he could find a listing of "possibilities" to solve actual puzzling cases. A complete listing of all the possibilities is impossible, but the "completeness" achieved must have been adequate from the beginning, because soon after publication, Tesch-

endorf's text became a classic, and has remained, to this day, with Alban Köhler's "borderlands" and Schinz's textbook, one of the basic triad of diagnostic reference books for German-speaking radiologists.

But time goes on, and even the so-called "internistic" diagnostic roentgenology (encompassing Assmann's old textbook) has snowballed into a domain too vast for mastery by any single individual. This has been implicitly admitted by having part (B), devoted to the heart shadow, entirely rewritten by a "specialist" (Dr. P. Turn), so that ample space could be given to cardiac catheterization, and in general to the correlation between form and function, *e.g.*, by comparison with special procedures, including both kymography and electrokymography. Parts (C), the aorta, (D) the esophagus, and (E) the diaphragm (the latter entitled very precisely: differential diagnosis of the roentgenologically visible alterations of the diaphragm), are by the master himself. A second volume, to (re)appear sometime in the future, contains the sections on abdomen.

When reaching for such a wide scope, minor discrepancies can never be completely avoided, and pointing them out would (in this case) serve no useful purpose, as it is sometimes done by reviewers who wish to prove they have read "the" book from cover to cover, when all the "professionals" know that careful perusal by a "connoisseur" will uncover in twenty minutes at least three well taken controversial points in most any publication. By comparison with previous editions, aside from the fundamental changes in part (B), almost all the reproductions in part (D) have been replaced. The typographic presentation is excellent in all respects, the numerous bibliographic indications encompass the universal literature (with predominance of German and English speaking authors) until 1957, and aside from individual preferences for the inclusion, or exclusion of, or emphasis upon, certain specific items, even the critical reader will have few, if any objections to the matter presented in such a palatable manner. Consequently, Teschendorf's new first volume is herewith highly recommended, both for its educational value, and as a reference guide to help in the solution of diagnostic problems.

E. R. N. Grigg, M.D.

Traitement chirurgical de la Recto-côlite Ulcéro-Hémorragique. By E. Delannoy, Professeur de Clinique chirurgicale à la Faculté de Médecine de Lille; and M. Martinot, Moniteur de Clinique chirurgicale à la Faculté de Médecine de Lille. Paper. Price, 1,400 fr. Pp. 120, with 26 illustrations. Masson et C^ie^, Éditeurs, 120 boulevard Saint-Germain, Paris 6^e^, France, 1957.

This monograph is based on the observation of a comparatively small number of patients, but each patient was well studied from the point of view of clinical course, medical and surgical management and ultimate result.

The conclusions reached by the authors are in general accord with those of other writers and may be summarized as follows: The forms of ulcerative colitis which do not respond to medical management or which interfere with work or social activity should be treated surgically. Appendicostomy, cecostomy or colostomy should not be employed because they are ineffective. Ileostomy may be employed to relieve acute symptoms but cannot be expected to achieve a cure unless the colon is also completely removed. The rectum may or may not be excised, depending on its condition and its response to medical management. When the rectum is to be removed male patients should recognize the danger of impotence associated with this operation. The authors present their personal technique for an anal ileostomy with capacity for retention of fecal material.

Of interest to Anglo-American readers is the statement that polyposis does not develop frequently in French patients affected by ulcerative colitis. When present, polyps are regarded as a sign of healing without great malignant possibilities.

Cesare Gianturco, M.D.

La Broncografia nella Fisiopatologia Bronchiale. By Dario Gandini and Giovanni Juliani. Cardboard. Price, L. 4,000. Pp. 200, with 141 illustrations. Edizioni Minerva Medica, Corso Bramante 83–85, Torino, Italy, 1957.

This monograph on bronchial physiology in health and disease follows another monograph published by D. Gandini on clinical bronchography in 1955.

In the first part the authors discuss the various techniques for the study of bronchial movements, including fluoroscopy, serial roentgenography, kymography and cinematography.

The second part is devoted to the study of

bronchial motions and contractions and to a discussion of mental and pharmaco-dynamic influences.

The third part endeavors to show the changes of bronchial physiology caused by asthma, emphysema, chronic bronchitis, bronchiectasis, infections, bronchial stenoses, tuberculosis, pneumoconiosis, and pleural diseases.

The techniques used by the authors are necessarily specialized and the results obtained may be difficult to interpret in clinical practice. However, this monograph is of value for those interested in the finer points of bronchial physiology.

Cesare Gianturco, M.D.

The Treatment of Fractures. Volume III. By Lorenz Böhler, M.D., Director of Accident Hospital, Vienna; Professor of Accident Surgery, University of Vienna. Translated from the Thirteenth German Edition by Alfred Wallner, M.D., Diplomate, American Board of Surgery, and Otto Russe, M.D., Director of the Accident Hospital, Vienna. Cloth. Price, $21.00. Pp. 2,307, with 1,699 illustrations. Grune & Stratton, Inc., 381 Fourth Avenue, New York 16, New York, 1958.

This volume completes the author's presentation of the methods employed by himself and his associates in the treatment of injuries. The recommendations made are based, as in the two sister volumes, upon an immense case load treated at the Accident Hospital in Vienna. This includes over 660,000 injury cases and his experiences in both World Wars and in Spain in 1937. All injuries involving the lower extremities from the lower femur distalward are its scope.

An organized plan of treatment for each specific injury is presented with meticulous attention to detail. The approach, the evaluation, the materials needed, their manner of utilization, follow-up care and end result statistics are related, often provocatively. The volume is easy to read; it is well indexed. The illustrations are excellent. An appendix dealing with the author's philosophy of the surgery of trauma is included. The need for preventative education and training is stressed.

This volume shares your reviewer's feelings for its predecessors. Many American and British surgeons will not agree with the author's methods in many instances. However, they cannot resist being stimulated by their presentation. The material used in the compilation of the three volumes represents a fantastically large amount of work by the author and his associates. Collectively they justly deserve to be classified as an encyclopedia of trauma. These volumes deserve a place in the library of every surgeon interested in traumatic injuries.

Frank P. Walsh, M.D.

Atlas d'Anatomie stéréotaxique. By J. Talairach, M. David, P. Tournoux, H. Corredor, and T. Kvasina. Cloth. Price, 10,000 fr. Pp. 91, with 99 illustrations. Masson et Cie, Éditeurs, 120 boulevard Saint-Germain, Paris 6e, France.

Stereotaxy is such a recent development that the term is not included in standard medical dictionaries prior to 1952. It is defined in the big Webster, and the literal translation of its etymologic components signifies arrangement or motion in space.

In neurosurgery, stereotaxy means the method by which definite points in the brain are located (and selectively destroyed, at a distance and with the least damage to intervening structures, through electro-coagulation or, still experimentally, with ultrasound waves) by reference both to superficial landmarks and to internal landmarks, provided through positive, negative, or double (roentgenographic) contrast procedures.

The imponderables of the method are the incomplete knowledge of the physio-pathogenetic relationship between certain structures in the brain and their precise function in man, paired with just as incomplete knowledge of the anatomic relationship between these structures and recognizable landmarks. Some authors contend that in practice highly accurate approaches are not always prerequisite for good results, and that less selective stereotaxic "destruction" is preferable, based on the frequently made observation that surgical annihilation causes much less functional damage than a spatially equivalent vascular accident. This is certainly no good reason why we should not accumulate an adequate knowledge of stereotaxic anatomy (spatial arrangement) of brain structures, because, sooner or later, it will enhance our chances to achieve more precise effects in stereotaxic surgery (spatial motion). To this end, the atlas of Talairach presents in a luxurious form (richly endowed by the publisher

with extra-heavy paper and life-size illustrations) the result of the correlated anatomo-roentgenographic studies, performed since 1949 by the senior author, in collaboration with his associates. It constitutes a substantial contribution to basic research in neuroradiology and neurosurgery, and is certainly a "must" in the reference library of all those institutions in which this phase of medicine is practiced.

E. R. N. Grigg, M.D.

Selektive Lungenangiographie in der präoperativen Diagnostik und in der inneren Klinik. By Prof. Dr. W. Bolt, Köln, Prof. Dr. W. Forssmann, Bad Kreuznach, and Landesmed.-Dir. Dr. H. Rink, Marienheide. Cloth. Price, DM 54.—. Pp. 200, with 205 illustrations. Georg Thieme, Herdweg 63, Stuttgart, Germany, 1957.

The authors describe their extensive experience, in over 2,000 cases, with selective angiography of the lungs. It is a method of studying the circulatory stream of the lungs, whereby an intracardiac catheter is directed into the hilar vessels and into a segmental and subsegmental pulmonary artery under fluoroscopic control, after which 5 to 10 cc. of contrast material is introduced into the catheter. Thus, one can study the visualized vascular stream of an isolated portion of the lung morphologically and functionally. Three filling phases are visualized: the arterial, capillary, and venous. The presence of the capillary phase indicates an intact alveolar structure. By this method it is possible to make a *regional* analysis of the physiologic and pathophysiologic conditions in the periphery of the vascular stream of the lungs, in which region the actual function of the lungs occurs. The method is employed only after other, more simple functional procedures and other tests have failed to clarify completely the underlying disease and especially when an operative intervention carrying considerable risk cannot be avoided. Selective bronchography is used routinely in differential diagnosis in the lung clinic and only when no definite conclusion can be reached is selective angiography of the lungs considered. The two methods may complement each other or may differ from each other. The more peripheral the process in the lungs, the more likely that it will be diagnosed by selective angiography in contrast to bronchography. There is an excellent historical review of the development of cardiovascular physiology. Reproductions of previously unpublished roentgenograms of Forssmann's original work with intracardiac catheterization are included.

The following conditions may be demonstrated by selective pulmonary angiography: (1) Vascular anomalies of the large veins and in the lung proper; (2) extent of vascular damage in lungs (obliteration of terminal blood vessels, absence of capillary phase and slowing down of blood stream), as an indication for surgical intervention such as segmental resection, lobectomy, or pneumonectomy; (3) visualization of the capillary phase permits differentiation between reversible and irreversible atelectasis; (4) the status of the lung vascular pattern may determine whether or not a collapsed lung may re-expand; (5) a differentiation may be made between an intra- and extra-pulmonary process; (6) it enables the visualization of the secondary pulmonary vascular sclerosis due to heart failure and may be of aid in establishing the indication for certain heart operations. During this procedure, venous and cardiac pressure measurements as well as blood gas analyses are done prior to the selective angiography. An activation of an inflammatory pulmonary process has not been experienced.

It is shown that severe changes in silicosis, by themselves, do not necessarily lead to an increase in pressure of the lesser circulation. Only when a marked emphysema accompanies the silicosis, does there result, in the advanced stages, an increase in pressure of the pulmonary artery. In early bronchogenic carcinoma, there is demonstrated a stretching and narrowing of the vascular bed, with a slowing down of the flow of the contrast material. Vascular changes in emphysema and in tuberculosis of the lungs (acute, miliary, and chronic) are discussed in detail, with numerous illustrative cases. In tuberculosis of the lungs, right heart hypertrophy occurs only when the extensive bilateral chronic process is accompanied by secondary reduction of the lung vascular stream and degeneration of the remaining functional lung parenchyma. Lung vascular changes in the presence of pneumothorax, after lung resection, decortication and thoracoplasty are demonstrated clearly. It can be predicted preoperatively whether or not decortication will improve the functional status of the lung, depending upon the appearance of the angiogram. Selective pulmonary angiography has been found of great aid in reaching a

decision as to the benefit to be expected after operation for mitral stenosis. The vascular changes in the lungs signifying hyalinization and sclerosis contraindicate such an operation, since such changes are irreversible. Similarly, the method may be of value in patent ductus arteriosus and in interventricular septal defect.

A new method of regional analysis of ventilation called isotope thoracography developed by Knipping *et al.*, is described briefly. The patient inhales a radioactive gas, xenon 133, having a half life of 5.27 days. This gas forms no chemical combination within the body and is completely exhaled within a few minutes, thus being entirely harmless to the patient. The distribution of this gas over various portions of the lungs is registered by a Geiger-Müller or a scintillating counter and the values inscribed as a curve. Since this method can detect localized lung disturbances as does selective pulmonary angiography, the two may be used as a complement to one another.

The book is illustrated profusely with excellent roentgenographic reproductions, which show clearly to the minutest detail the points emphasized in the text. A very large bibliography, international in scope, is included. The reviewer considers this book a classic of its type and recommends it to everyone who is interested in the study of the lungs from a physiologic, medical or surgical aspect.

SAMUEL RICHMAN, M.D.

BOOKS RECEIVED

EINFÜHRUNG IN DIE MESSTECHNIK DER KERNSTRAHLUNG UND DIE ANWENDUNG DER RADIOISOTOPE. By Prof. Dr. H. Fassbender, Erlangen. Plastic. Price, DM 37.50. Pp. 223, with 142 illustrations and 15 tables. Georg Thieme, Herdweg 63, Stuttgart, Germany, 1958.

COLLECTED PAPERS FROM KING GUSTAF V'S JUBILEE CLINIC. VOL. VII. The Radiumhemmet, The Radiopathology Institute, 1955–1956. Cardboard. Numerous papers with illustrations. The Radiumhemmet, Karolinska Sjukhuset, Stockholm, Sweden, 1958.

THE MEDICAL ASSISTANT; A GUIDEBOOK FOR THE NURSE, SECRETARY, AND TECHNICIAN IN THE DOCTOR'S OFFICE. By Miriam Bredow, Dean of Women, Eastern School for Physicians' Aides, New York. Cloth. Price, $7.50. Pp. 430, with numerous illustrations. McGraw-Hill Book Company, Inc., 330 West 42nd Street, New York 36, New York, 1958.

FRACTURES AND OTHER INJURIES. Edited by Edwin F. Cave, M.D. Cloth. Price, $28.00. Pp. 863, with 611 illustrations. Year Book Publishers, Inc., 200 E. Illinois Street, Chicago 11, Illinois, 1958.

ACTIONS CHIMIQUES ET BIOLOGIQUES DES RADIATIONS. Edited by M. Haissinsky. Third series. Cloth. Price, 3.600 fr. (Paper. Price, 2.800 fr.) Pp. 222, with numerous illustrations and tables. Masson et C^ie^, Éditeurs, 120 boulevard Saint-Germain, Paris 6^e^, France, 1958.

ACTIONS CHIMIQUES ET BIOLOGIQUES DES RADIATIONS. Edited by M. Haissinsky. Fourth series. Les Peroxydes Organiques en Radiobiologie. By R. Latarjet and others. Cloth. Price, 3.600 fr. (Paper, Price, 2.800 fr.) Pp. 156, with 49 illustrations. Masson et C^ie^, Éditeurs, 120 boulevard Saint-Germain, Paris 6^e^, France, 1958.

THE PSYCHOLOGY OF MEDICAL PRACTICE. By Marc H. Hollender, M.D., Professor and Chairman, Department of Psychiatry, State University of New York, Upstate Medical Center, and Director, Syracuse Psychiatric Hospital. Cloth. Price, $6.50. Pp. 276. W. B. Saunders Company, 218 W. Washington Square, Philadelphia 5, 1958.

AMÉLIORATION ET REPRODUCTION DES RADIOGRAPHIES PAR MODULATION ÉLECRONIQUE; LE LOGETRON. By A. Jutras, Professeur de Radiologie, Université de Montréal; and H. Fischgold, Neuro-Radiologist de l'Hôpital de la Pitié, Paris. Paper. Price, 4.500 fr. Pp. 122, with 16 illustrations. Masson et C^ie^, Éditeurs, 120 boulevard Saint-Germain, Paris 6^e^, France, 1958.

ACTION OF RADIATION ON TISSUES; AN INTRODUCTION TO RADIOTHERAPY. A Lacassagne, Chief of Service of Pasteur Institute, Director of the Radium Institute (Biological and Therapeutic Sections); and G. Gricouroff, Chief of the Laboratory of the Curie Foundation (Radium Institute); translated by Clarence C. Lushbaugh, M.D., and Gretchen R. Riese, M.S., Los Alamos Scientific Laboratory, University of California. Second edition. Cloth. Price, $6.25. Pp. 199, with 17 illustrations. Grune & Stratton, Inc., 381 Fourth Avenue, New York 16, New York, 1958.

INTERNATIONAL CONGRESS ON CLINICAL CHEMISTRY, STOCKHOLM, AUGUST 19–23, 1957; SUMMARIES OF INTRODUCTORY LECTURES AND ABSTRACTS OF INDIVIDUAL COMMUNICATIONS. The Scandinavian Journal of Clinical & Laboratory Investigation, Supplement 31. Paper. Price, Sw. Cr. 25. Pp. 325, with illustrations. Obtain through: AB Nordiska Bokhandeln (Medical Department), Drottninggat. 7, Stockholm, Sweden, 1957.

NIERAFWIJKINGEN NA PYELOGRAPHIE MET THOROTRAST (PATHOLOGICAL CHANGES IN THE KIDNEY FOLLOWING THOROTRAST PYELOGRAPHY). By Reinoldus Leonardus Oswald Marie Verhaak, Geboren te Zevenbergen. Paper. Pp. 156, with summary in English. Drukkerij Henri Bergmans N. V., Langenstraat 35, Tilburg, Netherlands, 1958.